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ROENTGEN KYMOGRAPHIC STUDIES OF ANEURYSMS AND MEDIASTINAL TUMORS*

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ROENTGEN kymography has been progressively developing since Sabat[10] conceived the original idea in 1911. A year later Gött and Rosenthal,[5] of Munich, devised a kymograph independently of Sabat's work and gave the name, "roentgen kymography" to the procedure. In 1916 Crane,[2] of Michigan, elaborated the method and was the first to apply it to a study of heart disease. Becker,[1] Knox,[8] and others, made contributions with the single-slit kymograph. To Stumpf,[15] of Munich, belongs the credit for originating multiple-slit kymography in 1928 and for perfecting the apparatus so that by 1931[16] the method was of clinical importance. Hirsch[6] introduced the method to this country in 1934.

We prefer the "step kymogram" to the "surface kymogram" because in the former each wave is the record of the same point on the border of the heart (Fig. 1).

The essential part of the kymograph is the grid. It is a sheet of lead in which narrow, horizontal slits are cut every 12 mm. apart. The opening in each slit is 0.4 mm. wide as advocated by Hirsch. During a single, continuous exposure of one second, the film moves slowly down behind the lead grid a distance slightly less than the spacing between two slits—actually 11 mm. The points on the border of the heart that are recorded are those which overlie a slit in the grid.

In the kymogram each frame is a record of the movements of a single point on the border of the heart inscribed on 11 mm. of film as it moves down behind the grid. The chambers of the heart and great vessels produce characteristic types of waves. This makes it possible to visualize accurately the position and extent of the various chambers and great vessels in the cardiac silhouette (Fig. 2). The lower leg of each wave is the record of diastole, and the upper leg, of systole.

By a simple measurement with calipers it is possible to determine the events that occur simultaneously in the various chambers of the heart and great vessels, since all points on any wave in any frame that are equally distant from the white line at the bottom of a frame, occurred at exactly the same moment. For instance, if the point of beginning left ventricular systole is 3 mm. above a white line and at a point 3 mm. above the white line in an aortic frame, the aorta is bulging outward as a result of ventricular contraction.

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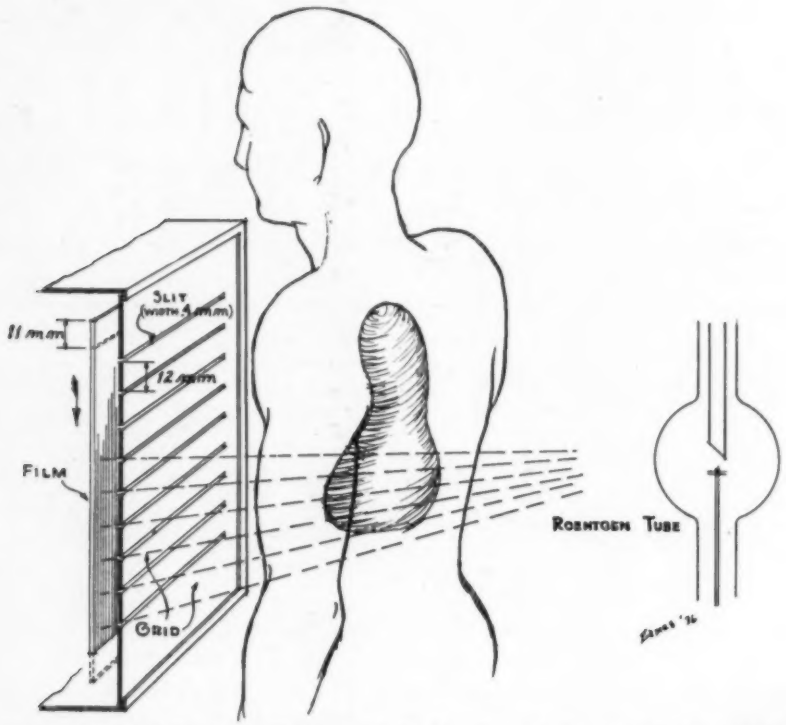


Fig. 1. *Diagrammatic Schema of Roentgen Kymograph.* In the "step kymogram" the grid is fixed—only the film moves. The grid is a large sheet of lead $\frac{1}{8}$" thick in which narrow, horizontal slits are cut 12 mm. apart. The opening of each slit is 0.4 mm. wide. During a single, continuous roentgen exposure of one second, the film is slowly moved down behind the grid a distance of 12 mm. Note that the small areas on the heart border to be recorded are those which overlie a slit in the grid.

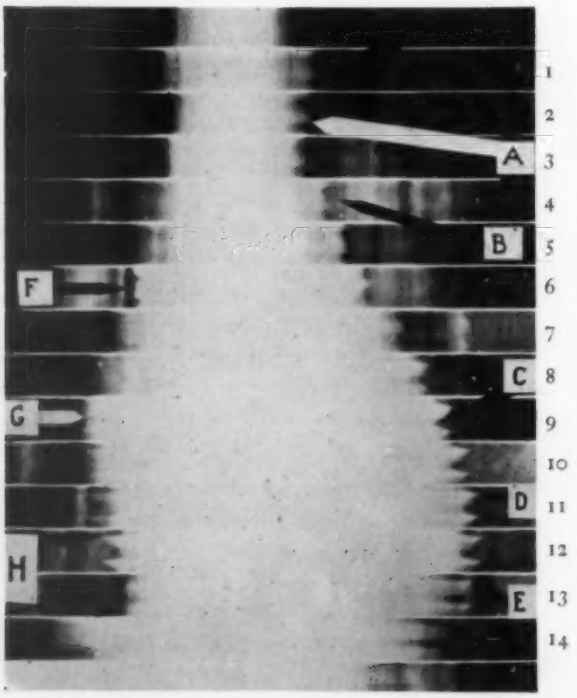
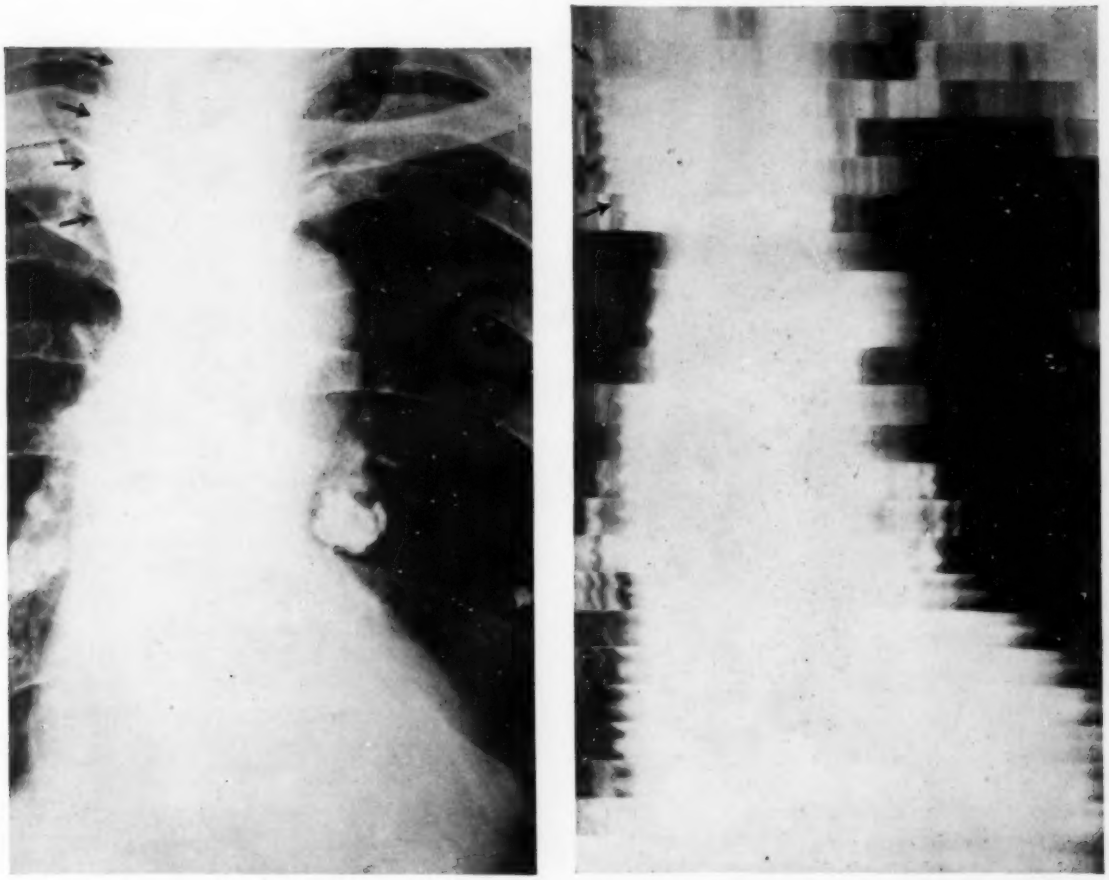


Fig. 2. *Roentgen Kymogram of Normal Heart.* Each frame (the space between two white lines) is the record of a single point 0.4 mm. wide on the border of the heart as it moves medially during systole and laterally during diastole. The trough of each movement wave is the position of the point at maximum systole, and the peak of the wave is the position of the same point at maximum diastole. The lower leg of each wave is the record of diastole and the upper leg, of systole. A single wave is the record of the change in position of a point on the border of the heart during one cardiac cycle. Fast movements are recorded as horizontal lines and slow movements, as oblique or rounded lines. *A*, peak of aortic wave attained just after onset of ventricular systole; *B*, wave of pulmonary artery; *C*, maximum ventricular systole; *D*, maximum ventricular diastole; *E*, beginning of auricular systole; *F*, wave of auricular appendage; *G*, beginning auricular systole; *H*, movement record of right ventricle.



A *B*

Fig. 3. *A*, conventional film of an aneurysm of the innominate artery. The convex shadow projecting to the right of the great vessels is very suggestive of an innominate aneurysm. Colored man, aged fifty-eight. Positive serology. Systolic murmur in neck with increased pulsation. *B*, roentgen kymogram of same patient. As anticipated, clear-cut vascular waves, aortic in contour and time occurrence, are recorded over the convex border of the aneurysm. Vascular pulsations, either expansile or transmitted, are usually present over the borders of an aneurysm and absent over the borders of a tumor. However, there are occasional exceptions to this rule.

The conventional chest film pictures the size and shape of the heart at an unknown moment in the cardiac cycle (Fig. 3*A*). These films are photographic "stills" of very animated organs and structures that are in constant motion, and in which absence of motion means death. The roentgenologist interprets them in terms of anatomical variations and pathological processes. There is only a faint suggestion of the physiological mechanism of the heart unless the patient is roentgenoscoped. Even then the movements are so small, rapid, and complex that it is almost impossible to analyze them in detail or to determine their time relationships.

The roentgen kymogram, on the other hand, records the size and shape of the heart in both systole and diastole, and, in addition, provides an accurate and objective roentgenoscopic record of the movements of the chambers and great vessels on a roentgenogram which can be studied at leisure and compared with subsequent kymograms. These films are graphic records of the physiological movements of the heart border, and are a means of detecting pathologic changes in such motion.

Because of these facts, it seemed that kymography should be an important aid in differentiating between aneurysms and intrathoracic tumors. Within certain limits,

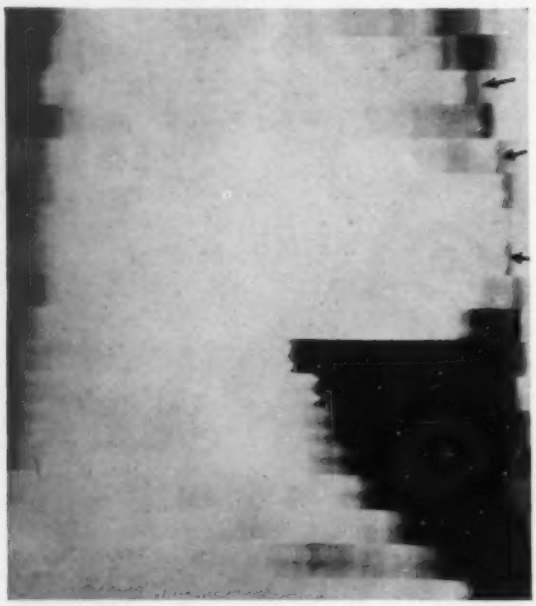


Fig. 4. *Roentgen Kymogram of Tumor with Transmitted Movement.* This man, aged twenty-one, complained of substernal pain and dyspnea for one year. Wassermann and Kahn tests were positive. At operation a large dermoid cyst was removed. The cyst lay directly against the heart and aorta which transmitted pulsation to the tumor. Thus tumors in close contact with the heart and great vessels may show transmitted motion.

it has proved to be helpful, but it is not infallible and must be carefully used.

Figure 3*A* is a conventional chest roentgenogram which reveals a heart moderately enlarged to the left with hypertrophy of the left ventricle. A convex, opaque shadow projects outward into the upper portion of the right lung field. The shape and position of the mass suggest that it is an aneurysm of the innominate artery.

The kymogram (Fig. 3*B*) of this patient shows clear-cut aortic movements over the borders of the convex shadow, a finding which strongly supports the clinical diagnosis of aneurysm.

Four patients with large, substernal thyroid glands and one patient with tuberculosis of an azygos lobe all possessed a shadow similar to that of an innominate artery aneurysm, but none of them exhibited movement at their borders.

These[13] and similar observations by others[3,4,7,11,16,17] substantiate the statement that most aneurysms display definite movement at their borders, either expansile or transmitted in origin, while most neoplastic tumors do not possess movement. There are frequent exceptions to this rule, and the correct use of kymography is of help in detecting them as is demonstrated by the following examples.

Case i. A man, aged twenty-one, had complained of increasing dyspnea, substernal pain and shortness of breath for the past year. His chest film revealed a large, rounded opacity filling the upper half of the left thorax. The Wassermann and Kahn tests were four plus. The kymogram showed definite, transmitted movement over the borders of the mass, favoring the diagnosis of an aneurysm (Fig. 4). No cardiac or vascular murmurs were heard. At operation a large, dermoid cyst was successfully removed. The cyst lay directly on the heart and aorta, which accounted for the vascular movement. Thus tumors in close proximity to the heart and great vessels frequently possess transmitted motion.

Case ii. Male, white, aged twenty-seven. A roentgenogram of his chest film revealed a large, rounded, opaque shadow in the upper and left portion of the thorax. A ring of calcific deposits encircled the mass. He complained of shortness of breath and substernal pain of four months' duration. No cardiac or vascular murmurs were audible. Wassermann and Kahn tests were negative. On the kymogram the borders of the mass were straight lines, indicating the absence of motion (Fig. 5). Our diagnosis was a mediastinal tumor, probably a dermoid cyst.

At operation the surgeon saw the tumor, felt it with his hands, aspirated it without result, incised it and was removing its contents when a sudden gush of blood identified the mass as an aneurysm. It was a rare, congenital aneurysm arising from the remnants of the ductus arteriosus. This experience epitomizes the difficulty in distinguishing between aneurysms and tumors and demonstrates that thick-walled aneurysms containing laminated clots may not possess even transmitted movements.

The presence of unquestionable expansile pulsation in a mass greatly increases its probability of being an aneurysm. The ac-

curate detection of such motion should be attempted in all cases in which there is a question of aneurysm.

Roentgen kymography offers the only objective and graphic method for detecting expansile pulsation in a tumor. Unfortunately, only a few aneurysms are so situated that they can be kymographed correctly. It is necessary that the kymographic grid be placed so that both sides of one diameter of the tumor are recorded through the same slit. If the movement is expansile, both sides of the aneurysm will move outward from the center of the mass at the same instant and medially at the same time. The diameter of the mass lengthens at systole and shortens at diastole. If the movement is transmitted, both borders of the mass shift in the same direction at the same time, and the diameter of the mass remains constant (Fig. 6, *A* and *B*).

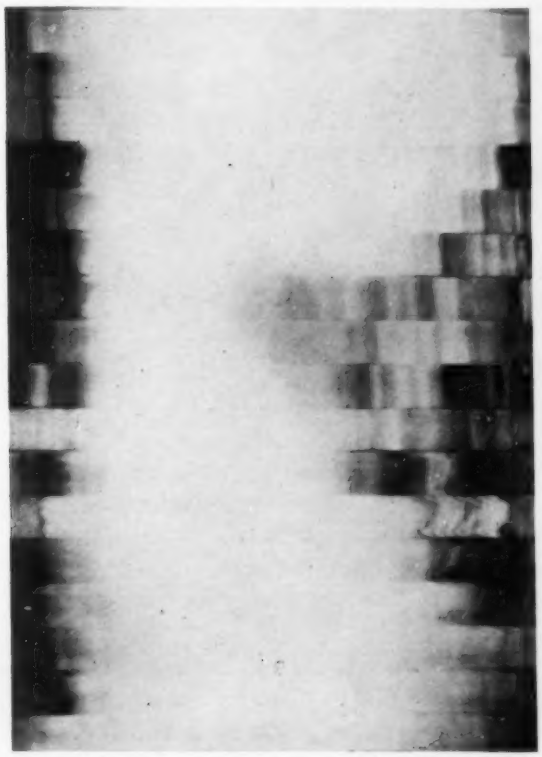


Fig. 5. *Roentgen Kymogram of Aneurysm Possessing No Movement.* Note the straight lines which form the border of the aneurysm. This film demonstrates that occasionally an aneurysm does not display motion over its borders.

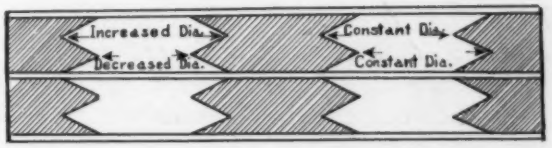


Fig. 6*A*. In the diagram of expansile pulsation both sides move outward at the same instant enlarging the diameter of the object. Likewise both sides move medially at the same instant decreasing the same diameter. In transmitted pulsation both sides of the object shift synchronously in the same direction and the diameter of the mass remains constant.

Serial kymograms of pulsating aneurysms made at bimonthly intervals can be used as a prognostic index for following the progress and course of an aneurysm. An

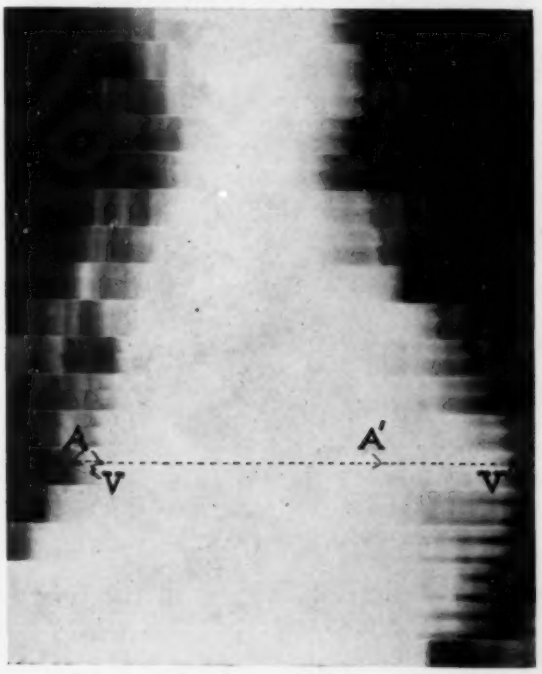
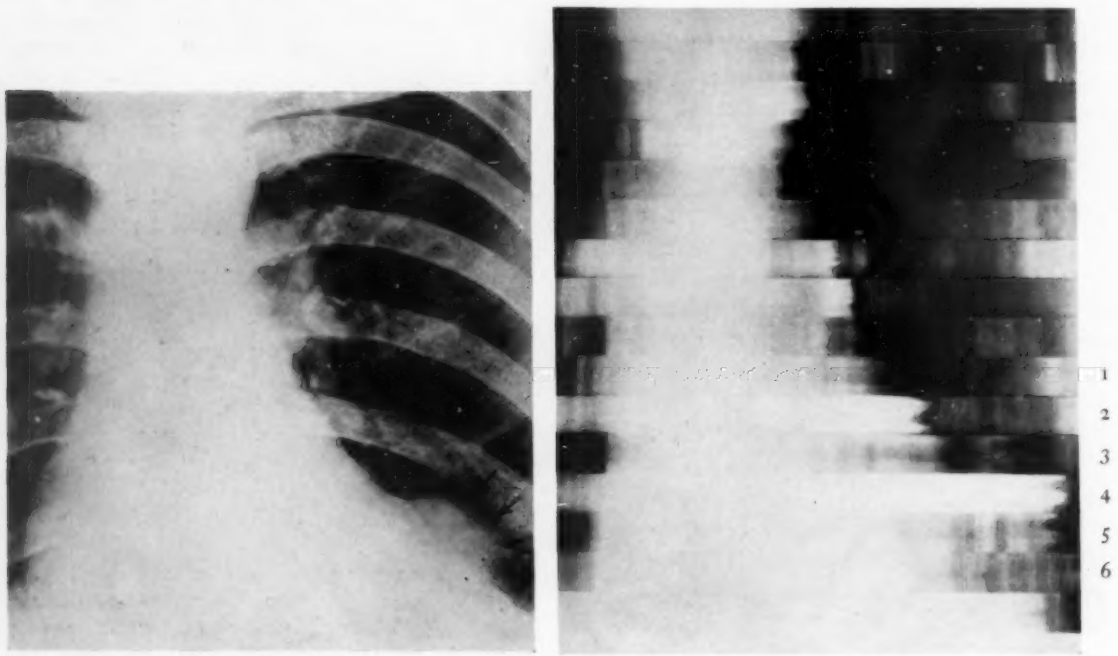


Fig. 6*B*. *Roentgen Kymogram of an Aneurysm of Descending Thoracic Aorta.* The aneurysm lies behind the heart shadow. Unfortunately its outlines are very faint in the print. On the film both sides (*A* and *A'*) of one diameter of the aneurysm bulge outward at the same instant denoting expansile pulsation. At that moment both ventricles (*V* and *V'*) are at systole. Thus the diameter of the aneurysm (*A* to *A'*) increases as a result of ventricular systole and decreases during diastole. Expansile pulsations can be detected only by recording both sides of one diameter simultaneously. Only a few aneurysms are so situated that expansile pulsation can be recorded.

increase in the extent of movement is interpreted as a thinning and weakening of the sac wall with the consequent risk of rupture. A decrease in the movement is interpreted as a thickening of the sac wall or blood clot which reduces the chances of rupture. In these aneurysms, the extent of movement is variable and depends on the volume of blood in the sac, the size of the clot, and the thickness of the sac wall. Only rarely is a tumor of sufficient vascularity to possess expansile motion.

Roentgen kymography provides another aid in the differentiation between aneurysms and neoplasms. This is the record of changes in the density of a mass which coincide with ventricular systole and diastole. At systole the density of an expansile aneurysm is greatest, and it is least at diastole. Such variations in density are frequently so slight that they cannot be detected by the unaided eye. Stumpf examines such variations in density with a densograph and has found them to be of considerable value. Millwee,[9] using his "scanograph" (a modified single-slit kymograph), has demonstrated astonishing variations in density coinciding with the heart beat.

Roentgen kymograms also have a Potter-Bucky grid effect which gives a better and clearer visualization of the thoracic aorta.



A *B*

Fig. 7. *Tumor of Pericardium. A*, conventional film of heart showing a convex shadow projecting outward from the lower border of the left ventricle. *B*, posteroanterior kymogram of same heart records the paradoxical movement over the border of the tumor (frames 4, 5, 6) as compared with movements of uninvolved ventricle in frames 1, 2, 3.

The differential diagnosis between aneurysms and tumors of the heart and pericardium presents many of the difficulties encountered in the recognition of thoracic aneurysms. In a previous publication[14] we reported a case (Fig. 7*A*) in which a convex mass continuous with the lower border of the left ventricle moved paradoxically to that of the upper and uninvolved portion of the ventricle.

The kymogram (Fig. 7*B*) records this alteration in movement very nicely. At the instant the upper and normal part of the

ventricle is in the medial position of systole, the border of the convex shadow is in the lateral position of diastole. Because of this paradoxical movement, we thought the protruding shadow was an aneurysm of the ventricle. Six weeks later the autopsy revealed the mass as a primary endothelioma of the pericardium which had invaded the left ventricular wall to fill its lumen. Thus a neoplasm of the heart may alter the movements in a manner similar to that produced by an aneurysm. The kymographic aids and pitfalls discussed for the diagnosis of thoracic aneurysms likewise apply to the study of shadows protruding from the heart border.

Our clinical evaluation of the roentgen kymographic method in general has been previously reported,[12] and its diagnostic value in the differentiation between aneurysms and tumors may be summarized as follows:

1. Roentgen kymography is helpful in the differential diagnosis between aneurysms and mediastinal tumors, but it is not infallible and cannot be employed successfully in all cases because:

a. Large, thick-walled aneurysms with laminated blood clots may not record motion.

b. Tumors in close apposition to the heart or great vessels may show transmitted movement at their borders.

c. An extremely vascular tumor may show expansile motion, although this is rare.

2. Roentgen kymography has demonstrated that aneurysms usually show vascular movement (either expansile or transmitted) at their borders while most tumors and inflammatory lesions do not.

3. Kymography provides a graphic method for distinguishing between expansile and transmitted motion if both sides of one diameter of a tumor can be kymographed simultaneously through the same slit.

4. The accurate detection of expansile movement in a mass strongly supports the diagnosis of aneurysm.

5. Kymography aids in the diagnosis of aneurysms by recording changes in their density coinciding with cardiac systole and diastole. Such changes are as significant as the detection of expansile pulsation.

6. Kymograms possess a Potter-Bucky grid effect which permits a better visualization of the course of the thoracic aorta with respect to adjacent masses.

7. Serial roentgen kymograms of aneurysms are of prognostic value in observing patients with aneurysms.

REFERENCES

1. Becker, T. Die Analyse des Elektrokardiogramms mittels der Röntgenkymographie. *Deutsches Arch. f. klin. Med.*, 1913–1914, *113*, 216–225.
2. Crane, A. W. Roentgenology of the heart. Am. J. Roentgenol., 1916, *3*, 513–524.
3. Faber, B., and Kjaergaard, H. X-ray kymograms of normal and pathological hearts. *Brit. J. Radiol.*, 1936, *9*, 335–344.
4. Gillies, C. L., and Kerr, H. D. Roentgen kymography of the heart and great vessels. Am. J. Roentgenol. & Rad. Therapy, 1936, *35*, 640–644.
5. Gött, T., and Rosenthal, J. Ueber ein Verfahren zur Darstellung der Herzbewegung mittels Röntgenstrahlen (Röntgenkymographie). *München. med. Wchnschr.*, 1912, *59*, 2033.
6. Hirsch, I. S. Recording of cardiac movements and sounds by the roentgen ray (kymophonoroentgenography). *Radiology*, 1934, *22*, 403.
7. Ihre, B. Röntgenkymography ad modum Stumpf as a method of examining the heart. *Acta radiol.*, 1934, *15*, 107–124.
8. Knox, R. Cardiac diagnoses; a survey of development of physical methods. *Proc. Roy. Soc. Med.*, (*Sect. Electroth.*), 1922–23, *16*, 1–30.
9. Millwee, R. H. Slit scanography. *Radiology*, 1937, *28*, 483–486.
10. Sabat, B. Ueber ein Verfahren der röntgenographischen Darstellung der Bewegungen des Zwerchfells, des Herzens, der Aorta. *Lwow. tygodn. lek.*, July, 1911, No. 28, 4. Republished in *Fortschr. a.d. Geb. d. Röntgenstrahlen*, 1913, *20*, 42–44.
11. Schilling, C. Die Anwendung der Flächenkymographie in der Diagnostik der Herzerkrankungen. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1933, *47*, 241–253.
12. Scott, W. G. Roentgen kymography. *Radiology*, 1937, *29*, 235.

13. Scott, W. G., and Moore, S. Roentgenkymography; its clinical and physiological value in study of heart disease. *Ann. Int. Med.*, 1936, *10*, 306–329.
14. Scott, W. G., Moore, S., and McCordock, H. A. Roentgen kymographic studies of cardiac conditions. *Radiology*, 1937, *28*, 196–210.
15. Stumpf, P. Die Gestaltänderung des schlagenden Herzens im Röntgenbild. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1928, *38*, 1055–1067.
16. Stumpf, P. Das röntgenographische Bewegungsbild und seine Anwendung (Flächenkymographie und Kymoscopie). (*Fortschr. a. d. Geb. d. Röntgenstrahlen*, Ergänzungsband 41.) Georg Thieme, Leipzig, 1931, pp. 1–78.
17. Wilke, A. Quoted by Stumpf, ref. 16.

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ROENTGEN KYMOGRAPHY OF THE NORMAL COLON

DEFECATION IN MAN*

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MOVEMENTS of the colon have been investigated through many channels of research. These include isolation of bowel (ileorectal anastomosis, with exteriorization of colon), abdominal window, balloons within the lumen, effects of drugs, section of the nerve control, various diets and the usual roentgen procedures. To the above we wish to suggest roentgen kymography of the active colon.

A brief consideration concerning past and present opinions relative to movements of the large bowel groups them thus: peristalsis, antiperistalsis, churning, pendular swing and systolic type movements such as found by Larson and Bargen[12] in the distal colon of dogs. Peristalsis and systolic contraction rings divide the adherence of most observers as the major factor in the propulsive mechanism of fecal transport. Peristalsis may be defined as a progressing wave of contraction following a wave of dilatation, the slow type of which is so clearly exemplified in the stomach. Systolic movements are local contractions in different areas, not progressing as a wave, but acting more or less as isolated constrictive and diastolic phenomena.

Before further discussion as to propulsion, it may be well to consider the function of the haustra. The three lines of thickening of the longitudinal muscle in the gut wall, called taeniae, act to decrease the length of colon, causing it to be drawn into folds or haustra, as noted by Kaestle, Gatewood,[7] and others, who found by roentgenoscopy in man that food is mixed and kneaded in the proximal colon by the alternate broadening and narrowing of the haustra. Barclay[3] in 1935 described his preliminary results with direct roentgen cinematography, taking films at the rate of one in five to ten seconds and recording: (*a*) the slowly changing outline of one of the haustra by superimposed tracings, and (*b*) the tracings of small opaque pills. He noted that the haustral movements are slow and intermittent and may be localized to individual haustra. They do not appear either to produce definite progression in the intestinal contents or to have directional intention. They seem to be concerned solely with the turning over of the bowel contents. Howell[10] and others concur that this haustral churning is non-propulsive and causes only local agitation. Local constriction of the circular fibers may be associated with shortening of the longitudinal fibers. As early as 1912, Kaestle concluded that the occurrence of deep haustration was physiologic, a point that seems to have been forgotten in the intervening years until its recent reappearance in the literature of so-called spastic colitis.

It is commonly accepted that the cecum is constantly active due to its churning of the liquid intestinal content, while the distal colon is normally quiet. Larson and Bargen found in dogs, by means of kymographic tracings with intraluminal balloons connected to a water manometer system, that when activity did take place in the distal colon, the most frequent movements were systolic pulsations and not peristaltic waves (which were rarely definitely identified). Cannon observed in 1902 that a deep constriction appeared near the advancing end of the feces coming from the proximal colon and almost separated a globular mass from the main portion. As the feces advanced, new tonic constrictions formed new globular masses. Thus these rings of constriction moved slowly from the cecum and propelled intestinal contents before them. Material was thus forced into the

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rectum by constriction, assisted by the abdominal muscles. Hertz in 1909 believed that contractions of the abdominal and diaphragmatic muscles aided to force feces into the rectum, with involuntary peristalsis starting back in the colon. Mass movements, as described by Hertz in 1907, by Holzknecht in 1909 and by many others, occur three or four times daily as the main propulsive movements from proximal to distal colon, possibly initiated by the entrance of food into the stomach as the so-called gastrocolic reflex. Some authors believe that they are caused by a powerful peristaltic wave but Holzknecht inclines to a tonic contraction of the circular coat. The haustral markings disappear and the fecal matter is formed into sausage-shaped masses which succeed each other progressing distally. Case saw them most often before and during defecation. One of us (R.A.R.) has many times found these mass movements after evacuation. Alvarez observed these contractions in patients with a tendency to diarrhea. Balli[2] noted seven spastic areas in the colon which he called points of origin for peristalsis and antiperistalsis.

The nerve mechanism involved has been extensively studied by Hatcher, Koppanyi,[11] and many others. It may be observed that the results of Bargen and Dixon[4] with studies of colostomy cases in man suggest that impulses for the above propulsive movements are transmitted by mesenteric nerves, in accordance with the theory of Alvarez[1] who advanced the idea of gradients of irritability in different regions in the bowel as the explanation of motor phenomena.

While the levator ani and other extrinsic anal muscles are important during defecation, the so-called valves of Houston, or plicae transversalis recti, which correspond to the lateral flexures of the rectum visible on the film have been thought by some writers to exert a spiral thrust against the anal sphincter. Pennington in 1900 advanced this theory and stated also that man is the only animal possessing these so-called valves and is the only animal that defecates regularly.

Conflicting opinions as to the rôle of distention of the rectum as the sole cause of defecation are to be gathered from the literature. Thus, Denny-Brown and Robertson,[5] in an investigation of the nervous control of defecation in man, held that the adequate stimulus is tension in the rectum, providing delivery of fecal material from the colon is sufficient. Garry[6] quotes the similar conclusions of Zimmerman (1909) and Hertz (1911) but disagrees that distention alone initiates defecation. He cites the clinical fact that tenesmus does occur in cases where there can be little or no distention of the rectum. A more convincing contribution by Hines, Lueth and Ivy,[9] who studied motility of the rectum in normal and in constipated human subjects by means of balloons in the rectum and sigmoid, demonstrated tonus rhythm and steep contractions as normal movements of the filled lower colon. They offer the logical opinion that the threshold for rectal sensation is raised in constipated cases. Hiller,[8] in describing the pathogenesis of anal fissure and fistula, held that, when the external sphincter is divided, the efficiency of the internal sphincter becomes reduced because the mechanical stimulus necessary for the maintenance of resistance to a dilating force is absent.

The observations which we are presenting from the results of our short series are advanced with the main idea of suggesting further possibilities for the study of large bowel physiology. Already the initial investigation of gastric kymography by Shilling,[13] Reinberg, Cramer and Pinke,[14] Stumpf *et al.* seems on a promising basis. As far as we have been able to determine, the current literature does not contain any reference to similar investigation of the large bowel. In our study, two methods of approach have been attempted: (*a*) roentgen kymographic films of the colon and rectum, before and during defecation, and (*b*) control examinations, using roentgenoscopy and films of the rectum during actual

defecation. The main objectives have been to note the changes in shape, position and the various phases of motility said to occur in the distal bowel. Of the 39 subjects used in our investigation 14 had screen study of defecation, 10 had films of the rectum before and during defecation without the use of the Potter-Bucky diaphragm, and 15 were kymographed. Opaque enemata were used, without any preliminary cleansing of the alimentary tract, and the effect of a semi-liquid stool apparently was thus obtained by mixing with the normal bowel material. Stools of this mixed type were observed in most cases. While a fully formed stool would be more desirable, it seems likely that even with a semi-liquid content of the colon diagnostic possibilities may be developed for kymography of the bowel.

The vertical kymograph was used to obtain exposures of the colon after injection of the barium enema. Varying periods of time up to twenty seconds were tried while the patient suspended respiration. The longer intervals were less satisfactory because of diaphragmatic tremors and body movement (as noted by the bone contour on the film). In order to protect the patient, no more than three kymographic films were taken on any one individual. Usually, after one film of the colon at rest, defecation was performed with the patient standing in the same position (with the legs slightly separated) before and during defecation. This exposure was limited to two seconds. *Obiter dicta*, it may be noted that the floor was protected, as no suitable receptacle was devised. Male patients of Kings County Hospital, under fifty years of age and having no abdominal symptoms, were the subjects. The cooperation of these volunteers was excellent, and to avoid any psychic effect on the results the examinations were conducted in as routine a manner as possible.

Details of the roentgenoscopic study are as follows: The regular bakelite top was removed from a horizontal roentgenoscopic table and a special fiber-board top substituted. This latter had an oval-shaped opening into which the patient placed his buttocks. An oilcloth funnel was attached below the opening and led to a bedpan supported on a wooden tray under the table top. After injection of the barium enema, roentgenoscopic notes were recorded in 14 subjects. Attention was directed to the intra-abdominal changes present when the patient was instructed to "bear down" as if to move the bowels but not actually to defecate at this time. Then evacuation was ordered and observed to its completion when possible. In some cases, a semi-recumbent position, with the back supported by a chair and pillow, was necessary for initiation of the act. By roentgenographing from below, films of the rectum at rest, in the phase of "bearing down," and during actual defecation were obtained in 10 other cases.

In interpretation of the kymograms (see Fig. 1), the time interval starts at the bottom of each frame. By noting the widening or narrowing of the bowel within each particular frame, activity is thought to be demonstrated. To examine the transverse colon and sigmoid area, the slits of the kymograph must be at right angles to those seen in Figure 1. Observations in the 15 cases that were kymographed indicate that at the onset of defecation no marked contractions are present (see Figs. 1 and 3). At this time the rectum is elongated and narrowed, and the anal sphincter dilated. Subsequent phases of defecation exhibit more obvious changes (see Figs. 4 and 5). As will be shown later, at the onset of defecation the rectum becomes elongated and narrowed, with relaxation of the anal sphincter. This seems to agree with the theory that colon contractions are instigated reflexly from the rectum.

The filled, non-defecating colon was found to be quiescent in most of the films. In a long exposure (20 seconds), tremors are transmitted from the diaphragm and body motion interferes with a proper evaluation (see Fig. 6). Mass movements, which occur three or four times daily, may

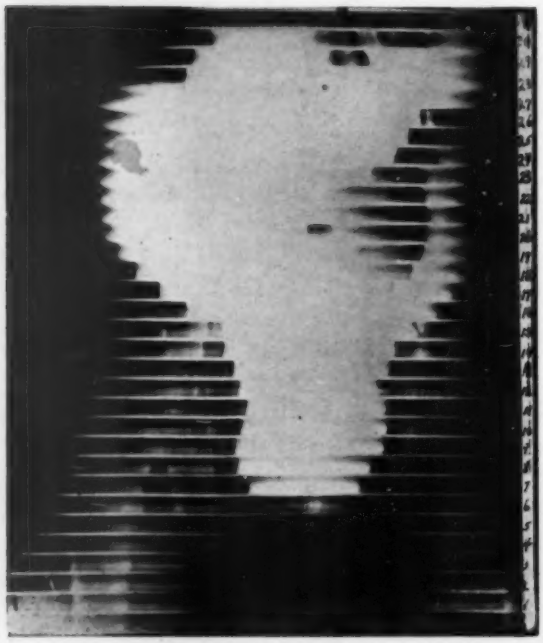

Fig. 1. Patient A. Filled colon at rest. Six-second exposure. Slight fibrillary changes of uppermost bowel, apparently transmitted from diaphragm. No filling or emptying present.

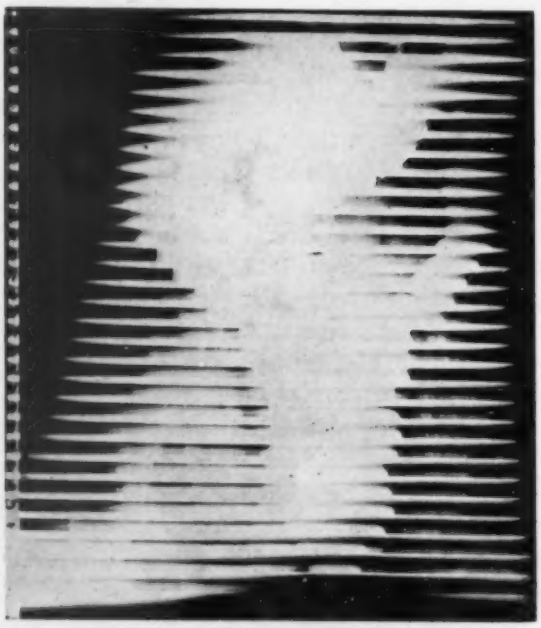

Fig. 2. Patient A. Onset of defecation. Two-second exposure. Descent of whole colon about 12 mm., due to increased intra-abdominal pressure. The rectum is longer and narrower. The sphincter is dilated. A thin stream of fecal content is being expelled. Note narrowing of bowel from bottom of each frame upward, in rectum and descending colon.

be demonstrable with the proper mixture of persistence and good fortune.

The following analysis of one set of kymograms of the colon before and during defecation (Figs. 1 and 2) is intended as a example to suggest one practical method of comparison. Later phases of evacuation will, of course, give more marked changes (Figs. 4 and 5). Analysis of Figure 1: The frames were numbered from 1 to 30, from below upward. During defecation there is a definite descent of the whole colon about the distance of one frame (12 mm.). This

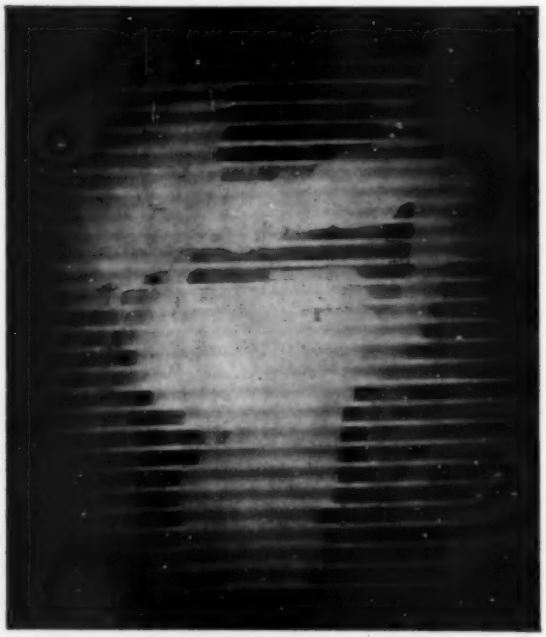

Fig. 3. Patient B. Onset of defecation. Two-second exposure. Sphincter dilated. Slight narrowing of bowel from bottom of frames upward in part of descending colon.

is ascertained by using the lesser trochanter as a base line for comparative level of bowel. The downward displacement is caused by increased intra-abdominal pressure as associated with contraction of the diaphragm and other voluntary muscles.

Note that before defecation the lowest point of the rectum is at frame 7. There are no out-thrusts to indicate filling or emptying. At the onset of defecation, the rectum is longer and narrower. There is dilatation in the region of the sphincter with a stream of fecal content being expelled in the lower

Table I

Rectum		Descending Colon	
Fig. 1 Before Defecation	Fig. 2 During Defecation	Fig. 1 Before Defecation	Fig. 2 During Defecation
Frame 7—7.0 cm.	7.7	Frame 17—4.0 cm.	3.3 cm.
Frame 8—8.3 cm.	8.5	Frame 16—3.6 cm.	2.7 cm.
Frame 9—9.3 cm.	8.3	Frame 19—4.0 cm.	3.0 cm.
Frame 10—9.3 cm.	8.4	Frame 20—3.7 cm.	3.2 cm.
Frame 11—9.0 cm.	8.3	Frame 21—2.8 cm.	2.7 cm.
Frame 12—8.5 cm.	9.8	Frame 22—3.1 cm.	3.3 cm.
Frame 13—9.8 cm.	10.8	Frame 23—4.1 cm.	

frames. Note the slight but definite narrowing of the bowel from the bottom of each frame upward, indicating contraction or emptying in the two-second interval. Besides elongation, the whole rectum is narrower than before.

Similarly in the descending colon, there is absence of contraction before evacuation. During defecation the bowel segments get narrower from the bottom of each frame upward, indicating contraction. A loop of transverse colon on the right that reached frame 30 before is now at frame 29, due to descent of the bowel for about 12 mm. For this reason comparative study must be done with a frame lower than before (see Table 1).

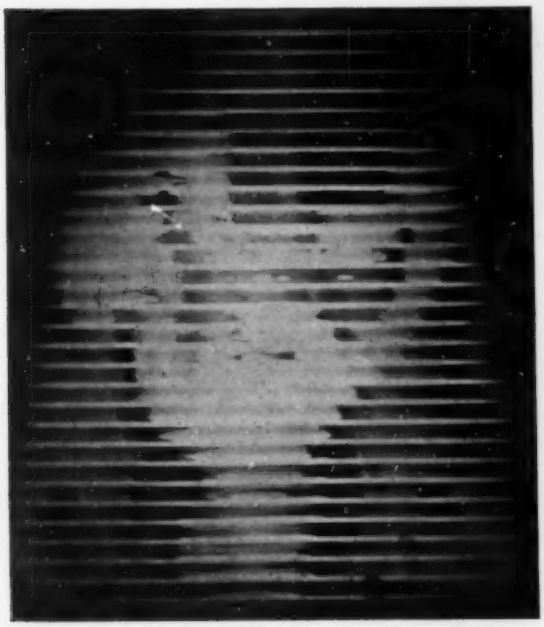

Fig. 4. Patient B. Two-second exposure. Further stage in defecation. Marked emptying of rectum. Descending colon contracting.

Fig. 5. Same series. Two-second exposure. Advanced stage of defecation. More contraction in descending colon. Some rectal frames completely empty before end of two-second exposure.

Note that in further stages of defecation (Figs. 4 and 5) more marked diminution in caliber of the bowel is present. In Figure 4, some of the frames in the rectum have emptied completely in less than two seconds.

Roentgenoscopic observations were made in 14 other normal cases as a check on the kymographic findings. The fecal content was also noted twenty-four hours

Fig. 6. Patient C. Twenty-second exposure of colon with respiration suspended. Not defecating. Most of the fine, needle-like out-thrusts are due to body motion, as seen by similar contour of the bone; others are fibrillations of the diaphragm. Deeper anal thrusts seem to be due to sphincter tonus.

after oral ingestion of barium and before the clysma. Voluntary contraction of the abdominal and diaphragmatic musculature caused a slight descent of the whole colon and rectum (about 12 mm.). Then the opaque enema was administered and enough injected to fill but not overdistend the gut. Thereupon the subject was instructed to defecate. Although there was evidence of increased intra-abdominal pressure, about half of the number were unable to evacuate until their position was changed to a semi-recumbent one. Inhibited or constipated subjects are not included. Upon actual defecation, motion was first observed near the lower end of the descending colon, where a slight "squeezing" of the bowel seemed to occur. Whether this occurred coincident with or immediately after relaxation of the anal sphincter was difficult to determine by roentgenoscopy. The rectum was noted to

Fig. 7. Fig. 8.

Fig. 7. Patient D. Filled rectum at rest. Sphincter closed. Plica transversalis recti present.

Fig. 8. Patient D. Demonstrates elongation of rectum during actual defecation. Plicae recti less deep.

be elongated. Upon continued defecation, mass movements were found in the left half of the transverse colon and in the upper half of the descending arm. The above method of evacuation continued until the transverse colon and distal bowel retained only traces of barium. In most of the cases, the ascending colon showed only slight emptying.

Films without the Potter-Bucky diaphragm, of the rectum at rest and with the patient defecating, disclose lengthening of the rectum (Figs. 7 and 8) by 3 or 4 cm. in a series of 10 additional cases. The plicae transversalis recti did not appear to play any important expulsive rôle. Even when the weight of the barium column was reversed (Trendelenburg), the plicae were present, which suggests that their function concerns intrinsic rectal tonus rather than an expulsive spiral thrust against the anal sphincter.

SUMMARY

(1) The act of defecation was studied in 39 male subjects having apparently normal colons; of these, 15 were kymographed. Control examinations with roentgenoscopy and films of the rectum were made.

(2) One practical method for comparisons of kymograms is exemplified. Preliminary observations are presented. The following conclusions should also be considered preliminary.

CONCLUSIONS

(1) The colon and rectum move downward about 12 mm. when increase of intra-abdominal pressure occurs.

(2) There is elongation and narrowing of the rectum during evacuation, with relaxation of the anal sphincter.

(3) At the very onset of defecation, the rectal and anal changes seem to precede motion in the colon, suggesting that colon evacuation may be stimulated reflexly from the rectum.

(4) When propulsion is definitely advanced, the contraction of segments of lower bowel varies irregularly with inactive segments. This segmental contraction of the circular fibers is shallow and seems to resemble a systolic and diastolic phase, rather than a peristaltic wave, as no definite progression of a wave pattern could be followed.

(5) The plicae transversalis recti are probably concerned with intrinsic rectal tonus rather than with an expulsive function.

(6) From a clinical aspect, further development of kymography of the colon would be desirable in the study of constipation. For example, comparison of the above points (type of contraction, elongation of the rectum, plicae recti, etc.) in normal and in constipated subjects may lead to useful information.

REFERENCES

1. Alvarez, W. C. Mechanics of the Digestive Tract. Second edition. 1929.
2. Balli, R. Sphincters of the colon. *Radiology*, 1929, *12*, 484–495.
3. Barclay, A. E. Direct x-ray cinematography with preliminary note on the nature of non-propulsive movements of large intestine. *Brit. J. Radiol.*, 1935, *8*, 652–657.
4. Bargen, J. A., and Dixon, C. F. Recent studies on physiology of human intestine. *Rev. Gastroenterol.*, 1936, *3*, 205–218.
5. Denny-Brown, D., and Robertson, E. G. Investigation of nervous control of defaecation. *Brain*, 1935, *58*, 256–310.
6. Garry, R. C. Emptying of lower bowel. *Tr. Royal Med.-Chir. Soc. Glasgow*, 1933–1934, pp. 1–8; in *Glasgow M. J.*, Nov., 1933.
7. Gatewood, W. E. Mechanics of the colon. *Northwest Med.*, 1930, *29*, 1–7.
8. Hiller, R. I. Anal sphincter and pathogenesis of anal fissure and fistula. *Surg., Gynec. & Obst.*, 1931, *52*, 921–940.
9. Hines, L. E., Lueth, H. C., and Ivy, A. C. Motility of rectum in normal and in constipated subjects. *Arch. Int. Med.*, 1929, *44*, 147–152.
10. Howell, Textbook of Physiology. Tenth edition. 1928.
11. Koppanyi, T. Studies on defecation, with special reference to medullary defecation center. *J. Lab. & Clin. Med.*, 1930, *16*, 225–238.
12. Larson, L. M., and Bargen, J. A. Physiology of colon. *Arch. Surg.*, 1933, *27*, 1–50.
13. Waters, C. A., and Kaplan, I. I., Editors. 1935 Year Book of Radiology. Year Book Publishers, Chicago, 1935.
14. Waters, C. A., and Kaplan, I. I., Editors. 1936 Year Book of Radiology. Year Book Publishers, Chicago, 1936.

MALIGNANT BRONCHIAL STENOSIS

BRONCHOGRAPHIC ASPECT*

By PEDRO L. FARIÑAS, M.D.
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THE great majority of bronchogenetic carcinomas are located in the first and second order bronchi, rarely in the third. These malignant tumors lead to a progressive bronchostenosis. The bronchographic sign of this condition, described somewhere as the "stop," indicates the complete obstruction of the bronchus and is, as we maintain, a late sign found in the advanced stage of the process (Fig. 1).

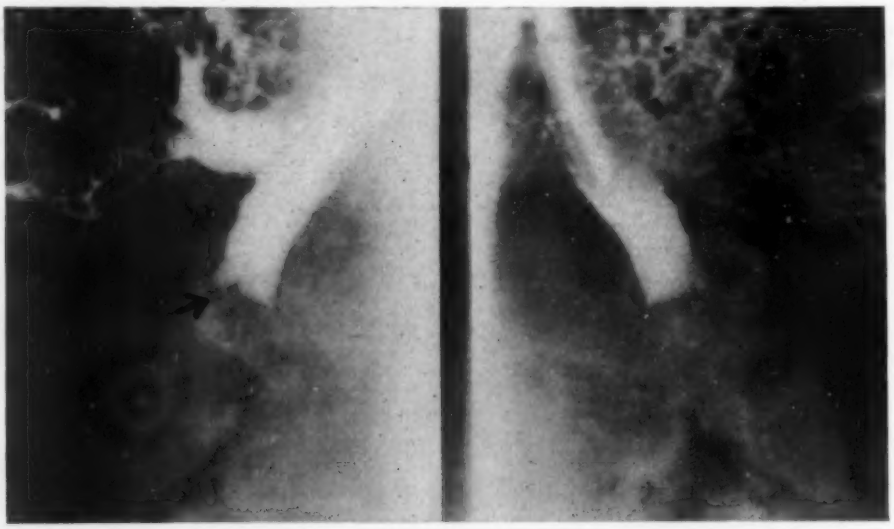

FIG. 1. Complete obstruction of the main right bronchus by advanced carcinoma. The "stop."

In 1933 we described the early signs of bronchogenetic carcinoma and here we will consider the malignant stenosis at its early stage; that is, before the complete obstruction of the bronchus.

The bronchographic aspect of the malignant stenosis varies with the type of the tumor. The polypoid type, growing towards the lumen of the bronchus, produces a well localized, more or less round or irregular stenosis with ragged borders, a characteristic sign of the malignant lesion.

The aspect in the bronchogram is of a filling defect in the form of a negative shadow that represents the displacement of the opaque oil by the tumor. Below the stenosis the bronchial dilatations are seen (Figs. 2*a* and *b*).

Benign polyp of the bronchus produces a similar image but the edges are regular and well defined (Fig. 3).

Very thick portions of mucus adherent to the bronchial wall may produce similar shadows, but they are generally multiple, changing their aspect at different moments during the examination, and bronchial dilatations are not seen below the stenosis as in the tumors.

The infiltrating type of tumors produces a concentric filling defect, fixed in aspect with irregular borders (Fig. 4).

When the lesion is far advanced, one has at first the impression of a complete stenosis and it is necessary to continue injecting the iodized oil during the roentgenoscopic examination in order to fill this narrow

* Read at the Fifth International Congress of Radiology, Chicago, Ill., Sept. 13–17, 1937.

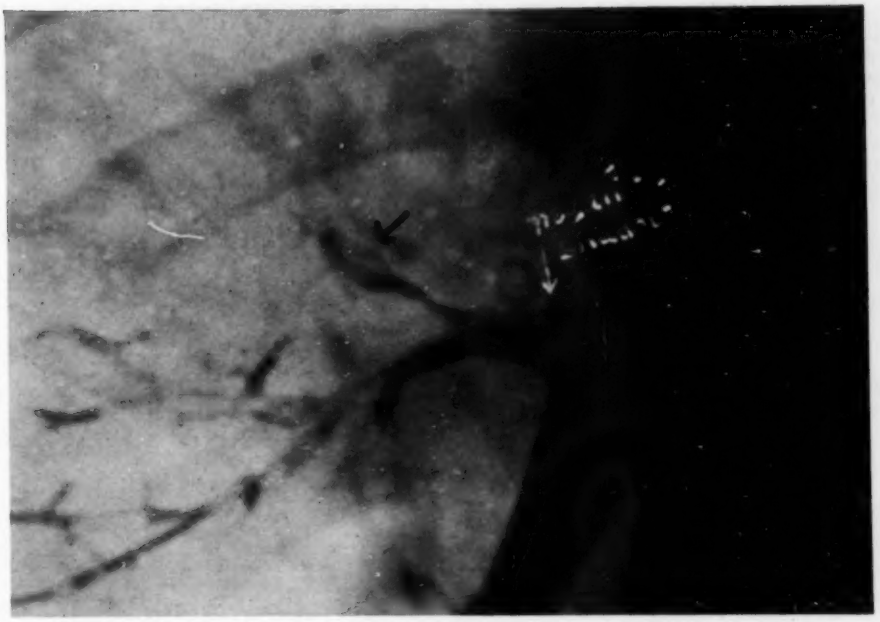

Fig. 2*a*. Polypoid carcinoma of the upper lobe bronchus. "Negative shadow" with irregular border.

Fig. 2*b*. Schematic drawing showing bronchographic aspect of the tumor.

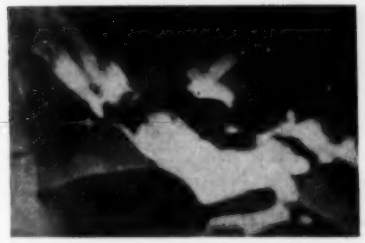

Fig. 3. Benign adenoma of the left main bronchus. Negative shadow with regular border. (From Peterson, H. O. Am. J. Roentgenol. & Rad. Therapy, 1936, *36*, 841.)

Fig. 4. Infiltrating carcinoma at the end of the right main bronchus. Concentric filling defect with irregular borders.

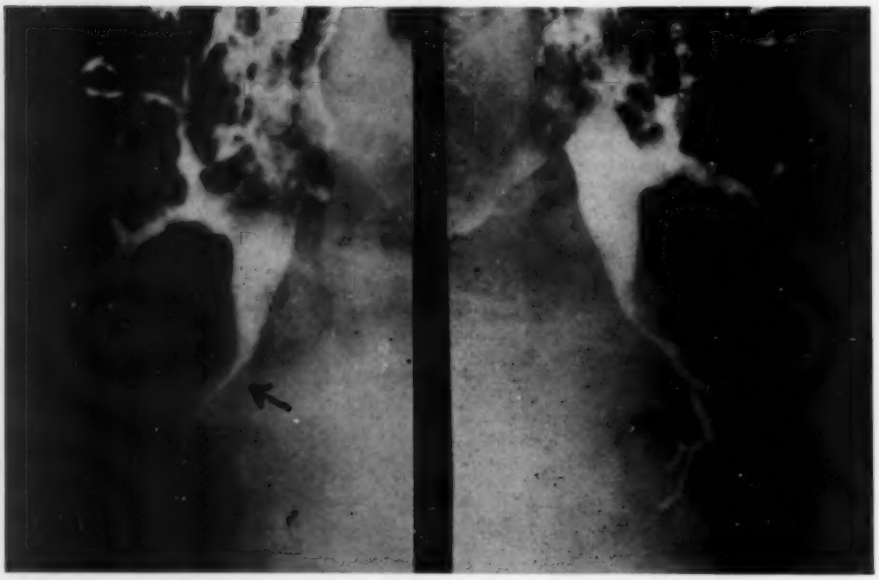

Fig. 5. Extensive carcinoma of the main right bronchus. Extended filling defect concentric in aspect.

portion; by this procedure a visualization of the incomplete stenosis is possible. Below the stenosis are always seen the bronchial dilatations (Fig. 5).

The inflammatory processes of the bronchial wall may cause shadows similar to those of the infiltrating type of tumor.

The draining bronchus of a pulmonary abscess shows an extended and irregular stenosis but, when closely observed, it is found to be quite different from the rigid aspect of the malignant infiltration. The pus and the iodized oil in the lumen of the bronchus give a mottled appearance to the narrowed portion.

The extended fibrosis of pulmonary ab-

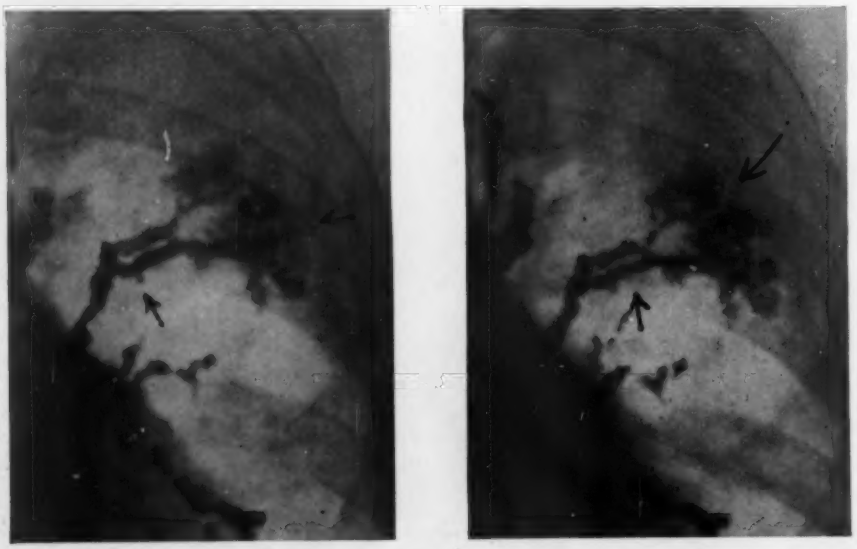

Fig. 6. Pulmonary abscess of the upper lobe. The cavity and the draining bronchus are filled. The draining bronchus shows a deformed and irregular mottled stenosis.

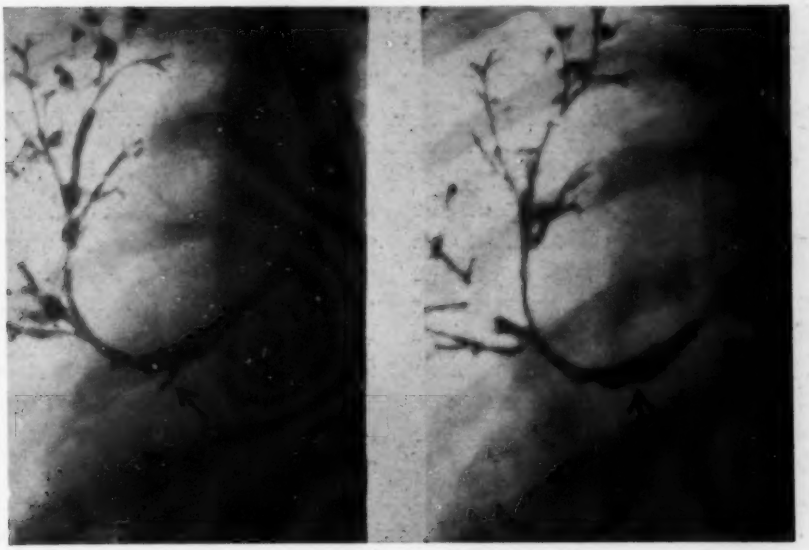

Fig. 7. Extended and irregular tuberculous bronchial stenosis of the main left bronchus with obstruction of the bronchus of the lower lobe.

scesses may displace the bronchial tree, thus giving rise to stenosis, but they are clearly differentiated from the malignant growths (Fig. 6).

The stenoses observed in bronchial tuberculosis are variable according to the stages of the process. When the disease produces an extended stenosis with irregular borders, it is very difficult and sometimes impossible to distinguish it from the malignant (Fig. 7). The clinical history, bronchoscopy and biopsy are of the greatest importance in differentiating the bronchial stenoses we have described, but when they are located in the third order bronchus of the lower lobes, or at the second or third order of the upper lobes, they may not be observed by the bronchoscopist.

REFERENCES

1. Fariñas, P., Castillo, P., and Basterrechea, C. Sobre el diagnóstico precoz del cáncer del pulmón. *Vida nueva*, May, 1932.
2. Fariñas, P. L. Serien-Bronchographien zur Frühdiagnose des Bronchialkarzinoms. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1933, *48*, 330–338.
3. Fariñas, P. L. Serial bronchography in early diagnosis of bronchial carcinoma. Am. J. Roentgenol. & Rad. Therapy, 1934, *32*, 757–762.
4. Piaggio Blanco, R., and García Capurro, F. La bronchografia. Montevideo, 1935.

THE SIGNIFICANCE OF LEFT AURICULAR DILATATION IN AURICULAR FIBRILLATION*

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THE determination of the size of the left auricle is often of considerable differential value in the study of the cardiodynamics and the etiology of a cardiac lesion. A study of its size in auricular fibrillation was undertaken to confirm the general impression that fibrillation has no direct relationship to the size of the auricle and to investigate the types of cardiac disease with chronic fibrillation which gave rise to dilatation.

METHOD OF STUDY

The hospital records of cases with auricular fibrillation in which a postmortem or a roentgen examination was recorded were selected for study. From the roentgen point of view, the left auricle was considered to be enlarged when there was posterior displacement of the esophagus. This was demonstrated best in the lateral view with the exposure being made while the patient was drinking a barium mixture and taking a deep inspiration at the instant of exposure.

A preliminary study of the esophagus was made in about 100 cases having no known cardiac lesion and including all variations in age, sex and shape of chest. In most cases the esophagus descended directly to the cardia of the stomach, except for the indentations caused by the aorta and left main bronchus (Evans[2]). In a few cases, there was a gentle posterior bowing from below the indentation caused by the bronchus down to the cardia. This bowing could often be straightened by deeper inspiration. Left auricular enlargement was most surely indicated by a sharp posterior displacement of the esophagus beginning immediately below the pulmonary artery indentation. This appearance was found when the left auricular dilation was marked and out of proportion to the size of the other chambers and particularly to the size of the left ventricle. When the left auricle was dilated moderately and in proportion to the left ventricular enlargement, as occurs in left-sided failure, the esophageal displacement was moderate and general.

It is conceded that this roentgen method of investigation of the left auricular size is not an accurate one. There seems to be no reason to doubt, for example, that a dilated left ventricle, enlarged posteriorly, will displace a normal left auricle with it. Esophageal displacement may be due to other conditions, as pericardial effusion, marked right auricular dilatation, adhesions of esophagus to the aorta, and extracardiac conditions. Nevertheless, with careful interpretation, fair accuracy is obtained and at present no other method lends itself so well to the routine investigation. Widening of the tracheal bifurcation, elevation of the left main bronchus and the appearance of the left auricular outline on the upper right cardiac contour are usually only confirmatory evidences of left auricular enlargement. Kymographic demonstration of auricular pulsation imposed upon the esophagus was not found to be of any great practical value.

MATERIAL FOR STUDY

Ninety-six cases of chronic auricular fibrillation were found to be satisfactory for study. Thirty were examined at post mortem. The analysis of these 96 cases showed a distribution in regard to age, sex and etiology similar to that noted in other reported series (Evans,[3] Friedlander and Levine,[5] Brown,[1] Schwartz and Weiss[9]).

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The present cases were divided as follows:

Ninety-six cases of chronic auricular fibrillation

A. With rheumatic valvular disease—44 (32 females, 12 males).

Age incidence	10–20	2 cases
	20–30	6 cases
	30–40	6 cases
	40–50	17 cases
	50–60	10 cases
	Over 60	3 cases

B. Without rheumatic valvular disease—52 cases (27 females, 25 males).

(a) Arteriosclerotic heart disease (with or without coronary artery thrombosis)	38 cases
(b) Coronary artery thrombosis	2 cases
(c) Syphilis	1 case
(d) Graves' disease	5 cases
(e) Hypertensive heart disease	5 cases
(f) Etiology undetermined	1 case

Age incidence	Under 40	2 cases
	40–50	8 cases
	50–60	12 cases
	Over 60	30 cases

RESULTS

In all of the cases of rheumatic heart disease with auricular fibrillation there were definite valvular disease and marked enlargement of the left auricle. Cases in which there was a predominant arteriosclerotic heart disease but in which microscopic evidence of an old rheumatic infection was demonstrated at post mortem were not included in this group.

Of the 52 non-rheumatic cases, 22 were found to have a normal sized left auricle (7 by post-mortem examination). These were divided as follows:

Arteriosclerotic heart disease	17
Coronary artery thrombosis	2
Graves' disease	3

The available reports did not permit any analysis of the duration of the fibrillation. The irregularity remained constant throughout the hospital stay except in a few cases in which the administration of quinidine caused a temporary restoration of normal rhythm. There were sufficient cases, however, to indicate that a fibrillation of long duration could occur without left auricular dilatation. On one of the post-mortem cases in this group, for example, the auricles had been fibrillating for six years before death.

In 24 cases, there was a slight to moderate left auricular enlargement which was proportional to the general enlargement of the heart. The left auricular enlargement was less marked than the left ventricular enlargement. In all of these cases a marked dilatation and hypertrophy of the left ventricle were found. There was also clinical or post-mortem evidence of cardiac failure. The 23 cases were divided as follows:

Arteriosclerotic heart disease	6
Arteriosclerotic heart disease with coronary sclerosis	12
Myocardial disease with practically normal coronary arteries	2
Luetic heart disease	2
Hypertensive heart disease	1

In 7 cases the left auricular enlargement was marked and out of proportion to the general enlargement of the heart. These are described in a little more detail.

Case I (No. 356987). M. G., female, aged sixty-two, was admitted with moderate cardiac failure. Roentgenoscopy showed a marked enlargement of the left auricle and right ventricle, the left ventricle being only moderately enlarged. The clinical diagnosis was coronary artery sclerosis and auricular fibrillation. The additional diagnosis of mitral stenosis was made partly because of the roentgenoscopic findings, partly on the history of chorea at the age of five. There were no murmurs.

Case II. (No. 390229). L. M., male, aged fifty-two, was admitted with moderate failure. A diagnosis of essential hypertension, arteriosclerotic heart disease, lues and auricular fibrillation was made. Roentgenoscopy revealed an enormous cardiac shadow with enlargement to the right and to the left and encroachment on the posterior mediastinum. This suggested marked enlargement of the left auricle. In view of the marked dilatation of all chambers, how-

ever, the propriety of including the case in this present group might be questioned.

Case III (No. 391628). B. L., female, aged sixty-two, gave a history of two years of cardiac insufficiency. A loud diastolic murmur was heard at the apex and was transmitted to the axilla. A short pulmonic murmur was heard. P2 was louder than A2. The clinical diagnosis was arteriosclerotic heart disease, hypertension, coronary artery sclerosis, mitral stenosis of arteriosclerotic origin and auricular fibrillation. Roentgenoscopy showed a marked enlargement of the left auricle with moderate enlargement of the heart to both left and right.

Case IV (No. 378126). A. P., male, aged sixty-six, gave a five-year history of cardiac insufficiency. There were signs of marked cardiac failure. Four years before the present admission roentgenoscopy showed only slight dilatation of the left ventricle but marked enlargement of the left auricle and right ventricle. A clinical diagnosis of arteriosclerotic heart disease, hypertension, coronary artery sclerosis, auricular fibrillation and uremia was made. The enlargement of the heart proximal to the mitral ring could not be explained.

Case V (No. 375121). F. R., male, aged fifty-one, entered with moderate cardiac decompensation. At the time of admission, he was also suffering from acute appendicitis. Roentgenoscopy showed a cardiac configuration which suggested mitral disease with marked left auricular dilatation and moderate left and right ventricular enlargement. There were no clinical data to confirm a possible diagnosis of mitral stenosis; the clinical diagnosis which appeared most likely was arteriosclerotic heart disease with coronary artery sclerosis and auricular fibrillation.

Case VI (No. 355691). E. S., male, aged sixty, was admitted with moderate cardiac failure. The clinical diagnosis was arteriosclerotic heart disease, auricular fibrillation and coronary thrombosis with myomalacia of the left ventricle. Roentgenoscopy showed a general enlargement of the heart but particularly of the left auricle and right ventricle.

Case VII (No. 355427). R. S., female, aged fifty, gave a history of cardiac insufficiency of sixteen months' duration. Roentgenogram showed dilatation of all chambers with mitralization.

DISCUSSION

A. Cases with Rheumatic Heart Disease. It is not surprising that all of the cases of rheumatic mitral disease with fibrillation showed very marked enlargement of the left auricle. Even in those cases reported in children (Schwartz and Weiss[9]) where the mitral disease could not have been of very long duration, marked enlargement of the left auricle was present. It must be remembered in this connection that the cause of left auricular enlargement in rheumatic mitral disease probably has two components: the mechanical effect of the mitral stenosis and insufficiency and the presence of diseased auricular myocardium due to the rheumatic infection. The fibrillation may be an expression of the diseased muscle.

B. Cases without Rheumatic Heart Disease. The evidence appears to indicate definitely that left auricular enlargement is not a constant finding in cases of chronic auricular fibrillation even when the irregularity is of several years' duration. It would seem to be true that the enlargement of this chamber when present is a part of the cardiac disease and that the fibrillation is not related to its dilatation. These results are in accord with scattered observations in the literature. Parkinson,[8] for example, says, "I have not recorded any enlargement within a few hours or even days after the onset (of paroxysmal auricular fibrillation or flutter) in the cases I have observed." He states, "In any patient with auricular fibrillation that has lasted long enough, the left auricle may be enlarged. In complete heart block it often looks prominent." Flaxman,[4] in studying the influence of auricular fibrillation on the course of hypertensive heart disease, noted that when the fibrillation preceded and precipitated congestive failure, early death occurred. On the other hand, when the fibrillation occurred after the congestive failure had set in (for any period from one

month to several years), it had no apparent influence on the course of the disease except in relation to the cause of death and the comparative absence of additional occurrences common in hypertensives. Magee and Smith,[6] in discussing auricular fibrillation in hyperthyroidism, comment that advanced age seems to be more important in producing fibrillation than does hyperthyroidism.

There are no pathologic changes common to all fibrillations. Mohler and Crawford[7] have pointed out that patients with mitral stenosis, with or without fibrillation, have the same degree of disease. Nevertheless, the superimposition of a fibrillation by excluding an efficient auricular systole exaggerates the tendency to auricular distention, even though the part played by auricular contraction normally in ventricular filling is probably small.

In all of our cases with left auricular enlargement, congestive failure was present to a greater or lesser degree; and where the enlargement was proportional to the general cardiac dilatation, it could be explained on the basis of this failure. To verify this point, a control series of cases with congestive failure but without fibrillation was studied, and entirely comparable degrees of left auricular enlargement were found. Furthermore, the study of a group of cases of hyperthyroidism yielded the impression that distinct enlargement of the left auricle was uniformly associated with fibrillation and congestive failure.

The present series also appeared to confirm M. G. Brown's[1] observations that acute coronary occlusion is rare in permanent fibrillation. In our cases of recent coronary occlusion with fibrillation, the heart, and in particular the left auricle, was not enlarged. They were not examined at the time of the acute episode.

The few cases with disproportionate left auricular enlargement in non-rheumatic heart disease were more difficult to interpret. In one, the possibility of an additional rheumatic mitral disease was indicated. In another case, arteriosclerotic mitral disease was suggested. There is no question that "mitralization" occurs and that the most likely explanation is that the dilatation of the left ventricle produces tension on the mitral cusps, producing in turn functional insufficiency. We have observed at post mortem such cases of arteriosclerotic heart disease which revealed large left auricles and dilatation of the left ventricles but which did not show sufficient evidence of mitral disease to account for them. In any event, the auricular fibrillation is probably of no direct importance in the production and degree of the auricular enlargement. Disease of the auricular muscle itself, however, is probably a factor.

CONCLUSIONS

1. Analysis of the records of 96 cases of auricular fibrillation which had been studied by adequate roentgenologic or postmortem examination confirmed the current impression that the size of the left auricle was not directly related to the presence of the fibrillation.

2. Where the left auricle was enlarged in the non-rheumatic group there was uniformly an accompanying congestive failure.

3. Disproportionate left auricular enlargement in the non-rheumatic group was usually to be accounted for either by "mitralization" or by myocardial disease.

4. Auricular fibrillation of many years' duration may occur without auricular enlargement.

5. Dilatation of the left auricle in hyperthyroidism is usually associated with auricular fibrillation and congestive failure.

REFERENCES

1. Brown, M. G. Relationship of coronary arteriosclerosis to auricular fibrillation with special reference to the term "arteriosclerotic heart disease." *New England J. Med.*, 1935, *212*, 963–967.

2. Evans, W. The Course of the Oesophagus in Health, and in Disease of the Heart and Great Vessels. Medical Research Council. His Majesty's Stationery Office, London, 1936.

3. Evans, W. A., Jr. Long-standing cases of auricular fibrillation with organic heart disease. *Ann. Int. Med.*, 1936, *9*, 1171–1177.
4. Flaxman, N. Auricular fibrillation; its influence on the course of hypertensive heart disease. *J. Am. M. Ass.*, 1937, *108*, 797–799.
5. Friedlander, R. D., and Levine, S. A. Auricular fibrillation and flutter without evidence of organic heart disease. *New England J. Med.*, 1934, *211*, 624–629.
6. Magee, H. R., and Smith, H. L. Auricular fibrillation in hyperthyroidism; influence of age. *Am. J. M. Sc.*, 1935, *189*, 683–690.
7. Mohler, H. K., and Crawford, B. L. Pathologic changes in the heart in auricular fibrillation. *Am. J. M. Sc.*, 1934, *187*, 171–176.
8. Parkinson, J. Enlargement of the heart. *Lancet*, 1936, *1*, 1337.
9. Schwartz, S. P., and Weiss, M. M. Auricular fibrillation in children; its relation to rheumatic heart disease. *Am. J. Dis. Child.*, 1928, *36*, 22–39.

VOL. 40, NO. 2

ROENTGENOLOGIC EXAMINATION OF THE NORMAL BREAST*

ITS EVALUATION IN DEMONSTRATING EARLY NEOPLASTIC CHANGES

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IF THE roentgen examination of the breast is to be of real aid to the clinician, it must reveal the presence of pathological changes in their earliest stages—before they can be detected by clinical methods; but even if it can only confirm the nature of clinically detected abnormalities of the breast, it would still be a very helpful diagnostic aid. A thorough familiarity with the roentgen appearance of the normal breast is obviously first necessary before detection of the earliest abnormal changes can be possible. To this end were undertaken the studies contained in this report. Examinations were made on 142 normal girls and women. Two examinations were made of each patient arranged so that one study was near the onset of menstruation and the other after an interval of fourteen days. The roentgen findings were analyzed in relation to the menstrual, pregnant and lactational history, age, height and weight, previous breast operations and anomalies or derangements of the endocrine system.

EMBRYOLOGY OF THE NORMAL BREASTS

Opinions regarding the derivation, structure and physiological processes of this organ are still controversial but some appreciation of the possible variations in its normal appearance may be gained from consideration of the normal differences due to race, constitution, heredity, age, sex, climate, habits and developmental irregularities due to glandular growth factors.

The generally accepted theory of a phylogenetic relationship to the sweat glands, classifying the breast as a "modified sweat gland," has recently been questioned again by Lustig[11] and Dawson.[3] The first visible trace of the organ is found in the milk streak, microscopically demonstrable in embryos at 4 mm., which consists of a diffuse area of many-layered epithelium. About the second month of fetal life, it develops into a protruding ridge visible to the naked eye, extending on either side from the axilla to the groin. Being of slower growth than the surrounding tissue, this ridge finally disappears, leaving at certain intervals along its course minute elevations consisting of circumscribed accumulations of epithelial cells which constitute the milk buds of future breasts. Although there may be a number of such buds, only the fourth pair go on to form the breasts, the others undergoing retrogression during the normal processes of growth. These buds gradually increase in size and the anlage flattens out eventually, forming a slight depression. The central portion cornifies and from the basal surface, epithelial buds project into the subcutaneous connective tissue. These epithelial buds are at first solid but later branch out and canalize. A series of atypical sweat glands form about the nipple as the first rudiments of the glands of Montgomery. Unstriped muscle tissue about the primary downgrowth contracts to form the nucleus for the future nipple.

POST-NATAL DEVELOPMENT

At birth one finds a slightly depressed rounded area over each pectoral region, in the center of which is a small rounded nodule. At this period there is no microscopically demonstrable difference between the male and female breasts. A simple fan-shaped system of ducts has been developed. For a few days after birth the breasts increase in size. This swelling subsides in

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three or four weeks and is caused by the secretion occurring at this time. It is generally agreed that this is a true secretion, but here, too, Dawson[4] differs, attributing its appearance to normal growth processes, rather than to secretion made possible by differentiation of specific tissue. At this stage the organ consists chiefly of connective tissue. No corpus adiposum is as yet demonstrable. The parenchyma grows very slowly by sending out new buds from the ducts, but there are no real alveoli throughout childhood. By the end of the fourth month the nipple has formed, although not yet extending above the surface of the skin. There may also occur a very slight increase in the size of the breast as a whole at this period.

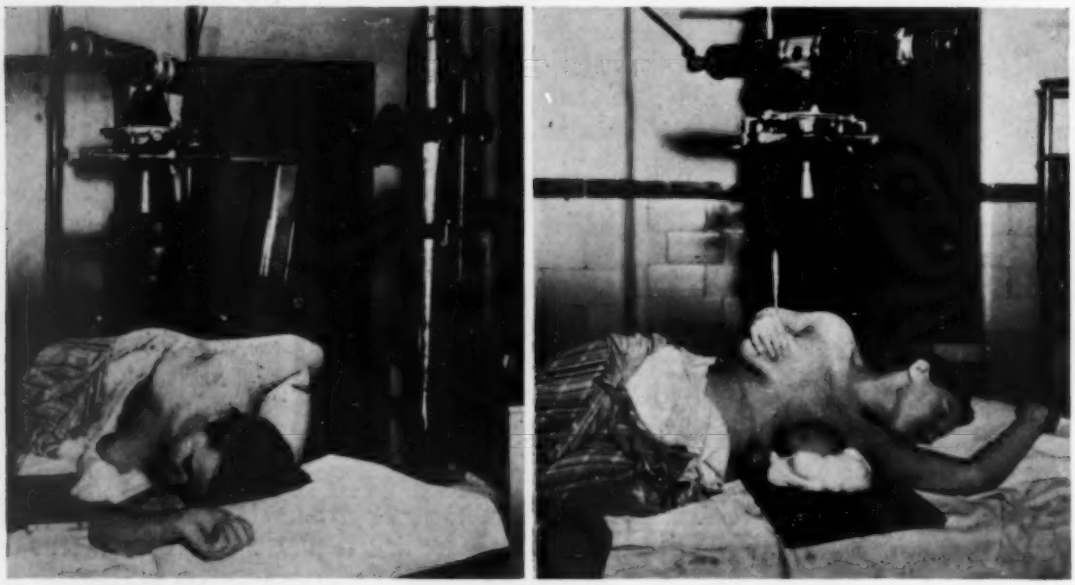

FIG. 1. Technique of examination. The patient is placed in the lateral recumbent position, the breast to be examined on the film, the other retracted with the homolateral hand. The breast may or may not be supported by absorbent cotton, but should extend out symmetrically from the chest wall. The central ray should be directed midway between nipple and chest wall, tangential to the chest and perpendicularly through the body of the breast. The factors that may be used are: 30–35 kv. (the higher kilovoltage for the compact virgin breast and the lower kilovoltage for the fat and atrophic breast of the adult and aged); 100 ma.; 0.3 second; 36 in. film target distance; patient immobilized—respiration suspended during exposure.

PUBERAL DEVELOPMENT

It is not until the onset of ovarian activity between the tenth and thirteenth years that the process of budding, branching and canalization is actively resumed, although no real growth of the corpus adiposum can be noted until the menses have been established for some time. Undue enlargement at this stage, like gynecomastia in the male, may be related to abnormally high secretion of estrin or theelin, possibly in turn related to pituitary or adrenal dysfunction. From the fourteenth to the sixteenth year the epithelial proliferating processes are particularly active. The lobules formed by the branching of the ducts are of varying size and gradually form in groups with simultaneous hyperplasia of the interlobular and periductal connective tissue[12] until the mature breast is developed. The size of the breast varies normally at all ages. *The actual size in no way indicates the stage of glandular development;* often it is due, however, to the variations in degree of fat content. The proliferating activity probably continues at a slower rate through the entire adult period

with coincident proliferation of the stroma and fatty tissue to form the normal hemispheric shape of the adult human breast. As has been stated above, the shape of the breast varies with race, climate and individual balance of endocrine function. The Caucasian and Mongolian races usually exhibit the hemispheric form, while in the darker races the breast often displays a shape resembling an udder. Individually the shape depends more or less on the consistency of the supporting tissue. If there is abundant adipose tissue, there results what is known as the hanging breast. The shape of the breast may be changed also by habits of dress such as the wearing of brassieres, etc. Also pregnancy and lactation tend to develop the hanging type of breast.

In boys, there is practically no growth from ten to twenty years of age. The male breast reaches its maturity between twenty and thirty years of age and begins to retrogress after thirty. Secretory activities may be observed at puberty in both sexes, giving rise to local swelling and pain.

ANATOMY OF ADULT BREAST

In the mature breast the organ is composed of glandular substance, fibrous tissue and fat. The lobes of the gland are divided into lobules separated by connective tissue and elastic walls. The nipple protrudes a little medial to the median line of the gland. Also the location of the nipple may be affected by the consistency of the mammary tissue. The organ extends from the parasternal to the anterior axillary line, and from the third to the seventh rib. In a state of rest the fat and connective tissue elements are in the foreground, glandular tissue being macroscopically visible only in the areolar area. In this state the connective tissue is frequently infiltrated with fat. The skin covering the breast is thin and firmly adherent, growing a little thicker toward the periphery of the breast. The areola surrounding the nipple may be elevated very slightly above the surrounding skin, and a fine groove is usually demonstrable between it and the base of the nipple. The skin covering the nipple and areola varies in color from pale pink to dark brown and becomes darker during pregnancy. Occasionally a secondary areola of paler hue has been noted. The small protrusions in the peripheral portion of the areola correspond to the glands of Montgomery. Beneath the skin is found fat covered with a thin layer of connective tissue, the latter radiating to the skin

Fig. 2. Section of a breast prepared by Dr. O. V. Batson, Department of Anatomy, Graduate School of Medicine, University of Pennsylvania; cleared by the Spalteholz method. Subject was a colored female, aged fifty-four, dyspituitary type. Section 2.5 cm. thick. Breast tissue very atrophic and fibrous septa and vascular network nicely shown.

and the pectoral fascia (Cooper's ligaments). The cutaneous connections are more rigid toward the peak of the breast, and less rigid at the base. The corpus fibrosum mammae within this fatty envelope also varies in size, covering the glandular parenchyma which is composed of fifteen to twenty separate glands each opening into the nipple. The whole construction of the breast is not unlike that of the lungs. The posterior surface of the breast is smooth, flat, sometimes concave. There may be more or less individual prolongations of the gland, usually axillary, rarely sternal. Each

FIG. 3. Roentgenograms of section of breast shown in Figure 2.

milk duct drains one lobe, receiving secondary excretory ducts from the lobules and acini. The ducts and acini are lined with epithelium on a layer of smooth muscle and are continuous with the epithelium of the body. The lobes are separated by a fibrous wall. The epithelium of the acini is formed of cylindrical cells resting on myo-epithelial or basket cells. The blood vessels and lymphatics penetrate into this connective tissue of the breast. The milk ducts are surrounded by a fairly thick connective-elastic tissue sheath.

The lymphatics spring from the lobules at the level of the acini and course along the milk ducts forming a subareolar plexus about the nipple, from which springs an infero-external branch, an infero-internal branch uniting to a trunk passing around the inner margin of the pectoralis major and terminating in the anterior group of axillary glands. A superior internal group passes directly to the subclavicular glands by way of the minor pectoralis. A less constant and less important internal branch penetrates deeper terminating in the internal mammary nodes. In some cases a lymph vessel from the deeper parts of the gland passes directly to the pectoralis major and clavipectoral aponeurosis terminating in the nodes of the subclavicular group. In many cases a branch has been observed passing directly to the supraclavicular nodes. The cutaneous lymphatics constitute a rich areolar network emptying into the axillary nodes, sub- and supraclavicular nodes and even into the nodes of the opposite side. These may descend to the abdominal wall uniting with the cutaneous lymph vessels of this region and of the upper thigh.

The mammary gland receives its blood supply from three sources. According to Schultz, the external half of the breast is supplied by two or three large branches of the lateral thoracic artery which springs from the axillary artery. The median and inner breast are served by branches of the internal mammary artery springing from the subclavian artery. Also branches from the third to the seventh intercostal arteries penetrate through the muscle and fascia to supply the breast. In the gland the arteries course along the ducts, anastomosing to form a network about the lobules, and sending tiny capillaries into the alveolar walls. The efferent veins follow the arteries. About the areola is a network of subcutaneous veins known as the mammary venous plexus. Muscle fibers appear in annular arrangement in the areola, while in the nipple the course of the fibers is axial from the base to the tip.

The cutaneous nerve supply of the breast is derived from the brachial plexus and intercostal nerves, converging toward the nipple which has an abundant supply of nerves often with tactile endings. The glandular nerves following the course of the ducts are derived

FIG. 4. Breasts in girl, aged thirteen. First menstrual period eight days prior to this examination. Breast tissue compact and roentgenographically undifferentiated.

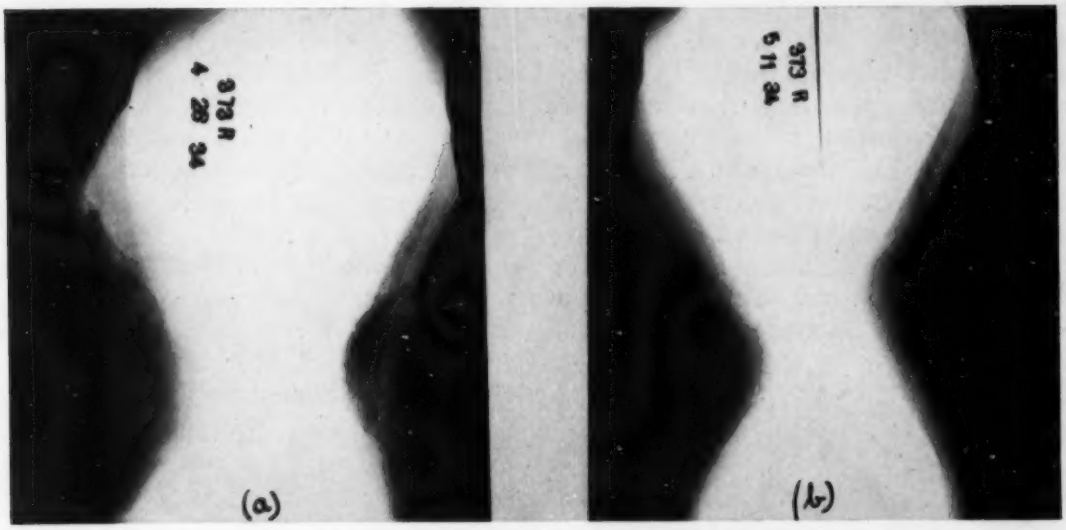

Fig. 5. Young adult breasts in woman, aged twenty. (*a*) breasts larger and less homogeneous two days before menstruation; (*b*) breasts small, more compact and homogeneous twelve days after menstruation. Menstrual history: 14(28/4-5).

from the fourth and sixth intercostal nerves. Sympathetic fibers are supposed to inhibit secretion, vagus fibers to stimulate it.

MENSTRUAL CHANGES

In women, the breasts undergo certain changes during the sexual cycle. Most theories agree that histological changes occur at the menstrual period, although Dawson could find no confirmation of such a process in his material. According to Rosenburg,[15] no lobules are present in the intermenstrual period but only milk ducts in a dense connective tissue stroma. The lobules are re-formed in the premenstrual period and undergo involution after the period. Dieckmann[5] believes that the lobule once formed persists and has observed them even in cases of prolonged amenorrhea. According to Dieckmann, menstruation does not cause epithelial activity but a fluid exchange in the lobular connective tissue leading to a premenstrual physiological edema around the lobule producing a loose tissue which disappears after the period, when the fibers become dense and collagenous. Dawson shares the above opinion, especially since he found lobules present in his material at all stages of the menstrual cycle and has seen them persist for years after the menopause.

At the menopause, natural or artificial, the mammary gland undergoes a marked involution. The glandular body atrophies, being reduced to a system of ducts encased in a fibrous sheath. The breast is formed almost wholly of fat, so that the organ itself may then appear to be increasing in size. The epithelium shows a tendency to proliferate with formation of cystic dilatations of the milk ducts. The double-layered epithelium of the milk ducts may also proliferate forming three or several layers of cells and multiple lumina or sometimes completely fill the milk ducts. Such epithelial proliferation is said to occur in 25 per cent of persons over forty years of age, especially in the peripheral portions of the gland. The epithelium of the acini will also occasionally proliferate with resulting cystic dilatations with more layers of epithelial lining than the usual single layer. The ordinary cuboidal epithelial cells change into huge cylindrical cells staining faintly with eosin very similar to the epithelium of the axillary sweat glands. These processes are closely related to chronic cystic mastitis. The supportive tissue of the breast also

undergoes senile changes. The collagenous connective tissue contains abundant blood vessels with thickened walls. The elastic tissue is increased around the larger milk ducts, and sometimes forms the main part of the stroma. The lumen of the milk ducts may be completely obliterated by connective tissue. In very old persons there may remain not even microscopically demonstrable glandular tissue. The involution of the different tissues varies in degree and

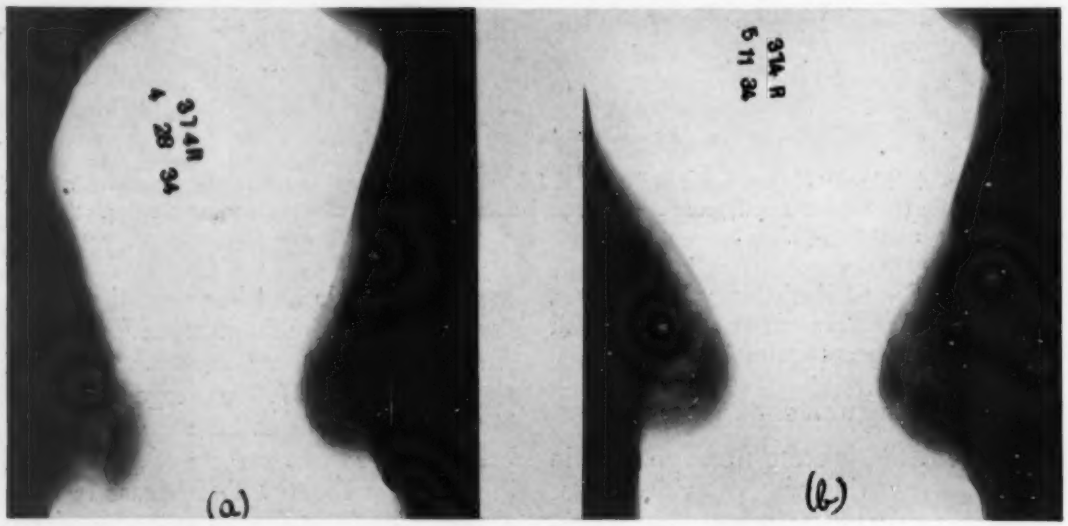

FIG. 6. Adult, single, aged twenty-eight. (*a*) seven days before menstruation, (*b*) seven days after menstruation. No appreciable difference is noted if slight differences of technique are discounted. Most of the breast parenchyma is subareolar, fat infiltrating in irregular digitations posteriorly. Menstrual history: 12(28/4-7), occasional dysmenorrhea.

also individually so that extremely old women may present breasts of normal appearance and considerably younger patients collapsed or wrinkled breasts. Calcification of arteries following the wake of arteriosclerosis may take place in irregularly distributed sections of the arterial tree.

TECHNIQUE OF ROENTGEN EXAMINATION

The roentgen examination may be made with the patient either in the erect or recumbent position; we prefer the latter. The patient is placed on the side with the arm closest to the table extended. The other upper limb is used to retract, laterally and backward, the other breast so that the central ray may be directed through the breast against the film without overlapping by the opposite breast. The patient may be rotated toward the prone position just sufficient to allow the breast to rest against the film with the nipple in profile. The central roentgen beam is then directed approximately perpendicular to the midsagittal plane of the breast itself and this is approximately tangential to the superficial plane of the pectoral muscle; the beam further traverses the breast midway between the nipple and the chest wall. This technique may be modified by allowing the patient to lie in the direct lateral prone position and the breast supported on sufficient absorbent cotton to extend it naturally away from the chest wall. This method has two disadvantages in that the examined breast is removed varying distances from the plate and when it is necessary to make repeated examinations of the breast, exact duplication of positions becomes rather difficult. It has the advantage of comfort to the patient and ease of centering the central roentgen beam. The small virginal breast with little fat infiltration will be found to require 3 to 5 more kilovolts

for satisfactory penetration than the much larger but fattier breast. Senile breasts, of course, will require 1 to 2 kilovolts less than the fatty adult breast. The average factors we employ are 32 kv., 100 ma., 0.3 sec., 36 in. distance and par speed screens.

THE ROENTGEN APPEARANCE OF THE NORMAL BREAST

The skin, subcutaneous fatty tissue, segments of the interlobar and interlobular connective tissue septa which are tangentially placed to the traversing roentgen rays, the fatty deposits in the stroma, the glandular tissue itself if sufficiently developed and so surrounded with the more radiolucent fat as to be demonstrable, the fascial cleft or space between the pectoral muscle and the corpus mammae can all be well demonstrated roentgenographically. It may require two or even more sets of films of different densities to demonstrate all these details. In the adolescent, small solid or firm virginal breast, with little fat infiltration, it requires an exposure with more penetrating rays to demonstrate the mammary architecture than in the much larger adult fatty breast. This increased penetration, though necessary to demonstrate the hyperplastic growing elements in the puberal or adolescent breast causes such an increased density in the film in the zone of skin as to make this region hardly perceptible. This means that a second exposure with roentgen rays of decreased penetrability is advisable to show the skin and subcutaneous area—and again, this exposure is usually insufficient to demonstrate details in the solid gland itself. For similar reasons, this duplication of examination is sometimes necessary for the adult gland because there is such a wide difference in the densities of tissue of the anterior or apical as contrasted with the posterior or basal portions of the gland.

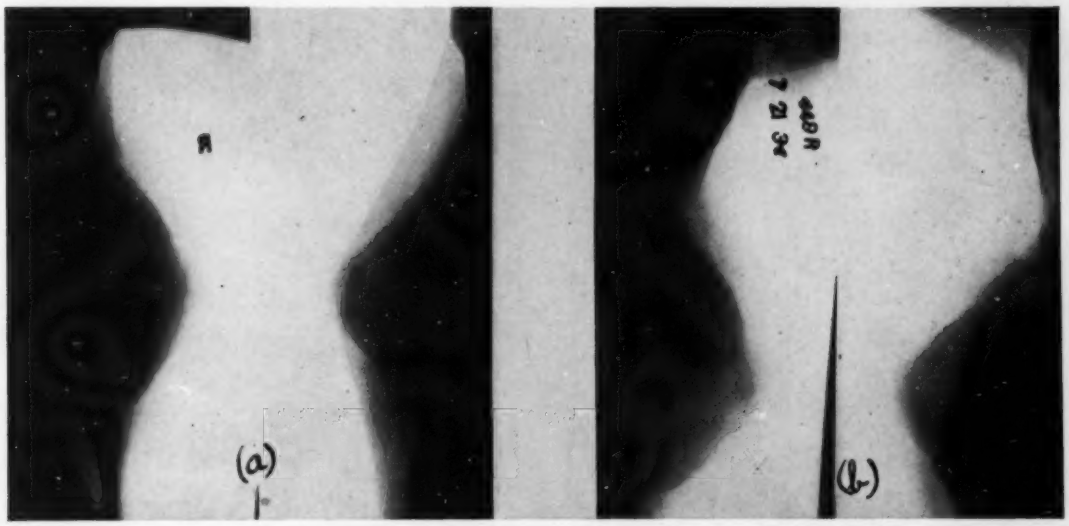

Fig. 7. Subject married, aged twenty-seven, no children. (*a*) eight days before and (*b*) at end of menstruation; breasts slightly larger. Tissues of corpus mammae are uniformly dense with practically no fat infiltration, much like a virginal adolescent breast of a thin subject. Menstrual history: 13(28/4-5), occasional dysmenorrhea.

There is still some controversy concerning the histological changes that take place in the breast during the menstrual cycle, but whatever they may be, they are not definitely or consistently demonstrable roentgenographically with the technique of examination used in this study. In this respect we seem to differ with Espaillat.[7] He has given an excellent description of the roentgen appearance of the normal breast and further describes menstrual

cyclic changes in serial roentgen examinations at eight-day intervals which we were unable to duplicate. The pre- and post-menstrual changes as seen in our roentgenograms would tend to bear out the conception of a pre-menstrual physiologic edema in the stroma, a view entertained by such investigators as Dawson, Dieckmann and Maximow and noted roentgenographically by Lockwood and Stewart[10] and Pendergrass.[13] In some cases, there seemed to be a slight increase in density with a slight decrease in the sharpness of detail of the fatty constituents of the breast during the four- or five-day period preceding menstruation. These changes, however, were certainly not pronounced enough to warrant limitation of roentgen examination to the remainder of the menstrual cycle for fear of obtaining blurred detail.

At puberty, there is such a wide variation in the size and shape of the gland already discernible that one can almost foretell with certainty the type of gland that will finally develop, characteristic for that given individual. This is especially true if the menstrual cycle has been established for a year or two. The young virginal breast is usually found quite firm, anchored to the chest wall so that it is difficult to bring the lateral aspect of the breast in contact with the film and still properly direct the central beam of roentgen rays. The distance between the breast and the film varies and the sharpness of roentgen detail varies proportionately. If little fat has invaded the stroma, the homogeneity of the projected shadow makes differential study of the component elements of the gland impossible. Fat alone casts the only normal contrasting shadow. It may be seen to invade downward from the subcutaneous layer or upward from the base, or it may be uniformly distributed. Since the fat alone is the contrasting medium for the stroma and ductal system and since this normally varies greatly in amount and character, it seems quite unreasonable that the roentgen studies could give us any information concerning such changes as benign epitheliosis and adenosis or so-called desquamative epithelial hyperplasia, changes that may make their appearance at this age. If these abnormal conditions are advanced, then, of course, the roent-

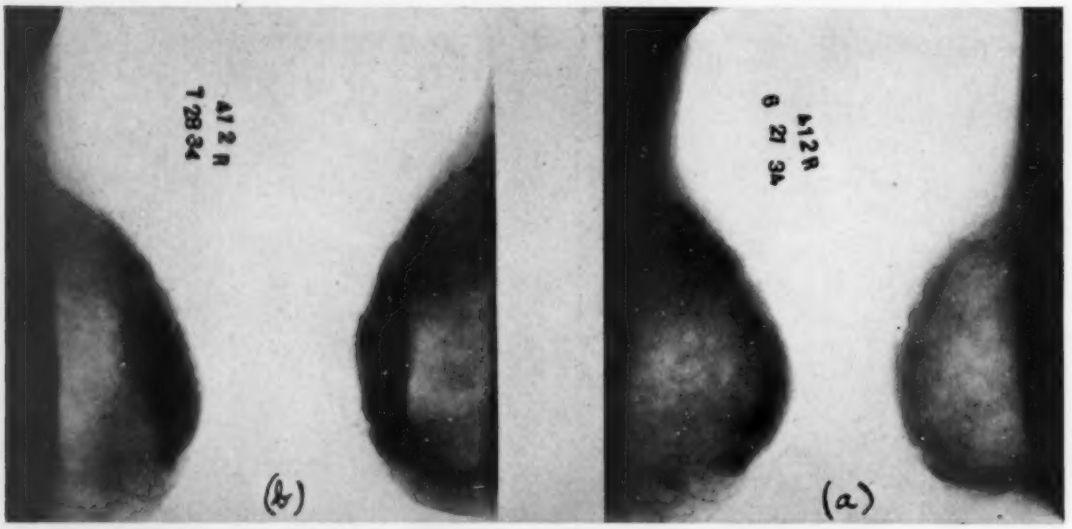

FIG. 8. (*a*) Breasts six weeks after lactation period, and eight days before menstruation in subject thirty-two years old. Two children, youngest ten months old. (*b*) Note progress of post-lactational involution, examination made at end of menstrual period.

genograms might confirm a clinical suspicion, but our findings would incline us to extreme caution in this direction. Fortunately, most tumors that appear at this age are benign, like the adenoma or fibroadenoma or so-called eccyclomastoma of Oliver and Major,[12] and if such tumors develop in a breast with liberal fat infiltration, their demonstration in the roentgenogram might be possible.

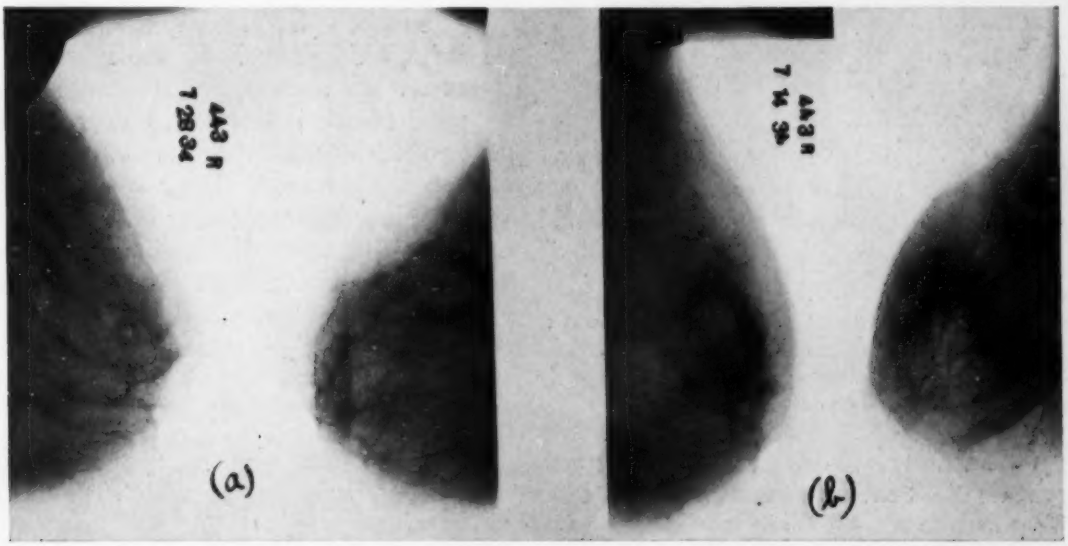

Fig. 9. Subject fat, married, aged thirty-two, 2 children, youngest eight years old. Both children nursed full time. Menstrual history: 14(28/4-5), slight pain in breasts before menstruation. (*a*) eight days before menstruation; (*b*) six days after menstruation when breasts seem still slightly fuller than at first examination. The complexity of the stromal striae and irregularity of fat deposit would make diagnosis of early metaplastic or neoplastic change very hazardous. Right nipple is retracted.

THE ADOLESCENT VIRGINAL BREAST

Practically all that was said of the breast at puberty in the preceding chapter pertains to the breast of this age period. If the individual shows a tendency to general or localized subcutaneous fatty deposits, the breast also is found to be similarly infiltrated. This aids considerably in throwing the glandular or fibrous connective stromal structures into relief in a more or less characteristic manner, depending principally on the distribution of the fat. The nipple, areola and subareolar area are practically never fat invaded so that in this area, a homogeneous shadow is present continuous with and of practically the same density as the skin. Diverging in more or less fan-shaped trunks and striae from this area are the ducts and stromal tissues. If the fat is sparse and deposed in islets, then a honeycombed appearance is produced in the roentgen picture of the corpus; if it is more abundantly distributed, there will be visualized irregularly spaced gutters or digitations or clefts of radiolucent fat between the other glandular structures and if the fat is excessive, an almost uniform radiolucent gland is demonstrated through which comparatively delicately formed stroma and ductal striations radiate irregularly, which in a general way converge toward the areolar area. Even the development of abnormal proliferations of stroma or epithelial tissues which may occur when these tissues are precocious in their growth cannot be demonstrated if these tissues are compact and are not contrasted by infiltrating fat. With normally such a wide variation in the degree of coarseness or fineness of the fibrous connective tissue and epithelial elements, and with such a difference in distribution of fat, it is hard

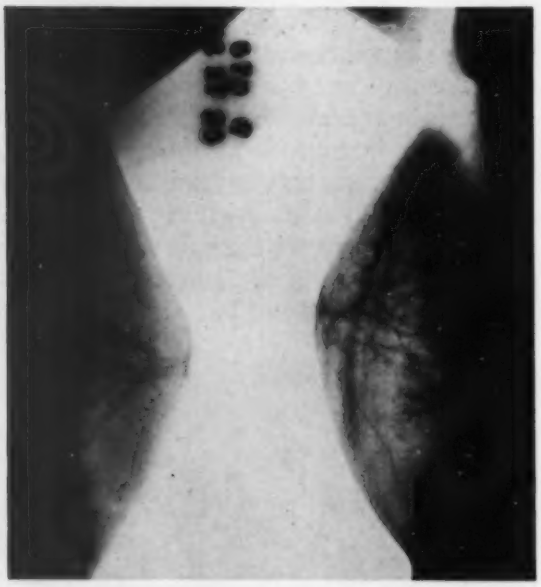

FIG. 10. Breasts in a subject forty-six years old, four months after menopause. One child—twelve years old—nursed nine months. Menstrual history: 13 (13/4–5). Atrophy of breast tissue moderately advanced with less complexity of stromal striae than in Figure 9.

to understand how a reliable roentgen diagnosis of Cheatle's[2] mazoplasia or Dawson's adenosis can be made.

THE ADULT BREAST (NON-LACTATIONAL)

The differences between this breast and that of the adolescent are usually very little. Modification in size and shape because of developmental characteristics is practically no longer operative. Only the continued repetition of the menstrual cycle causes an increased complexity in the architecture of the glandular structures within the gland itself. This results in the visualization of a few more radiating radicals of ductal and stromal tissues spreading out from the areolar apex to the base, since only those are clearly demonstrated that fall in the plane of the tangential rays; these radiating trunks are never seen as continuous prolongation but as detached segments of varying and changing thickness, criss-crossing each other because of overlapping projection of those in other planes. Principally because of these technical limitations, it becomes practically impossible to diagnose early abnormal proliferation in or around a single duct or a small group of ducts, or in a single or even in a few lobules so that an accurate roentgen diagnosis would seem unlikely at a stage before it can be made by the palpating hand or other clinical methods. This does not mean that such an accomplishment is impossible. As a matter of fact, it may be more easily accomplished than expected, particularly if by closer cooperation between the surgeon and radiologist an opportunity is offered to study roentgenographically all breasts suspected of undergoing abnormal changes.

There is a rather striking increase in the amount of non-fatty tissue in the mamma in the months immediately after lactation. The margins of the glandular tissue are poorly defined, the density reveals a wide range of variation and the entire picture

FIG. 11. Fat senile breasts in a woman aged fifty-one. The superficial veins and calcified arteriosclerotic arteries stand out prominently. The right nipple is retracted and the subareolar breast parenchyma is atrophic and densely fibrotic, the corpus being composed chiefly of fat. Patient had 10 children, the youngest twelve years old. Each was nursed about ten months. Menstrual history: 16 (28/3–4). Menopause six years ago.

of the breast is like that of one out of focus because of the slight burring of the structural detail. Only the superficial veins, especially those nearest the film, stand out sharply. Rarely a lymph gland or two can also be seen in the axilla, again depending much on its closeness to the film. As the post-lactational period lengthens, the stromal and parenchymatous elements undergo definite evidence of atrophy and retrogression, the architecture of which becomes sharper and more delicately etched-like in appearance. There is much variation in the rate of these involutionary changes, sometimes being very rapid in a period of a few months and at other times so slow as to require many years, extending even into the post-climacteric stage. It is familiarity with the vagaries of these changes that would seem essential before the slightest success could be obtained with the roentgen diagnosis of neoplastic diseases and the differentiation between benign and malignant neoplasia. The complex branching of the duct and ductule systems with their endings into clusters of acini, forming myriads of lobules increasing in each successive diverging plane toward the base of the gland, causes such an overlapping and confusion of shadows in the roentgenograms as to make practically impossible study of any particular or isolated portion of the mamma, especially near its base. Stereoscopy may be of some additional help. If serial studies are made at monthly intervals, it is possible that some fortuitous early diagnosis of neoplasia can then be made. The infiltrative branching, or radiating margins of a discovered neoplastic process might be regarded as indicative of malignancy, but one will have difficulty in distinguishing these ramifications from overlapping adjacent segments of interlobular and interlobar connective tissue and ducts. It is difficult to agree with Vogel[18] and Salomon[16] as to the relative ease of diagnosing neoplastic tumors in this type of breast and besides this, a very malignant process may have perfectly

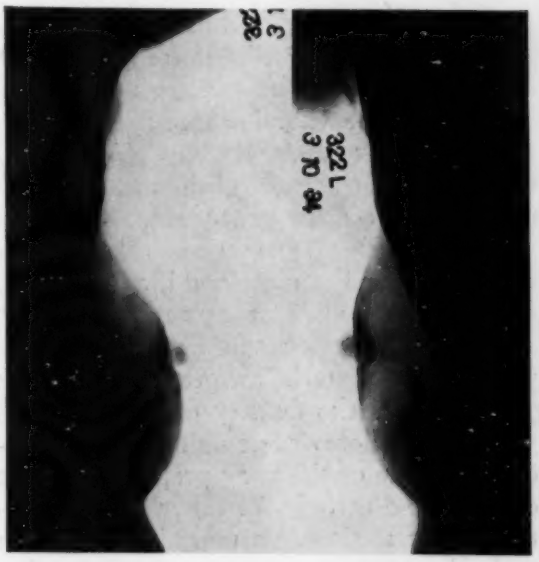

Fig. 12. Senile breasts in thin married subject, aged sixty-three, eighteen years after menopause. Nipples slightly retracted. Atrophic fibrous involution of breast tissue seen in faint striae radiating almost directly backward from the areolar area. Fat gives an almost homogeneous appearance to the corpus, making possible easy discernment of neoplasia.

smooth margins due to whorl-like growth, the resisting pressure by adjacent tissue causing pseudo-encapsulation. On the other hand, strange tissue architecture construed as neoplastic can be regarded as malignant if it overlaps the boundaries in or adjacent to the breast; that is, if it is seen to invade the corpus and subcutaneous fat and glandular stroma, or to extend backward into or beyond the submammary pectoral fascial cleft. The differentiation between a cystic tumor, a solid sharply defined neoplasm and a malignant growth with pseudo-encapsulation does not seem consistently possible at this time if one is familiar with the wide variation of morphology visible in the normal breast.

THE POST-CLIMACTERIC AND SENILE BREAST

At this stage, atrophy of gland parenchyma of different grades is present and the bulk of the mamma is fat. A few short

fine strands of atrophied parenchyma may be seen irregularly distributed (although still in a general fan-shaped manner) in the gland. The vascular tree is frequently better visualized in this gland than at any other age period. Sclerotic arteries with calcification are frequently seen. The diagnosis of new tissue formation in these mammae is relatively simple and if a decision of benignancy or malignancy can be reached, the chances for successful diagnosis of such pathological material are greater at this age period. Since the density of the breast at this time is more or less uniformly radiolucent because of the very preponderant fat content and atrophy of other gland structures, any radiopaque mass is easily seen and should excite diagnostic reasoning much as advocated by Seabold.[17] But his claim of demonstrable axillary nodes in the roentgenograms signifying probable malignant metastasis seems untenable to us. We have occasionally seen nodes in our series of normal individuals and we can see no theoretical reason why an inflamed node should not be as radiopaque as one which is the seat of malignancy.

COMMENTS

Changes and additions to roentgen technique might materially advance diagnosis of early neoplastic changes. Kymography, multiple examinations of the same breast in many planes with short film-target distance, and multiple serial examinations, preferably stereoscopic as done by Fray and Warren[8] and Ritvo, Butler and O'Neil,[14] of a breast suspected as the seat of an abnormal growing process are some techniques already receiving wide attention. Special devices like a wooden sling and curved tunnel with a small glass cup and rubber pipet to fix and hold out the nipple as recommended by Bianchini[1] deserve trial and improvement. Certainly, considerable improvement or development of the roentgen method of examination will be necessary before it can be regarded as a superseding diagnostic aid in the early stages of breast pathology. On the other hand, our present limitations are not so great as to preclude enthusiastic application of this method. There is no doubt that in certain instances, possibly more frequently than appears likely, diagnosis of early inflammatory and neoplastic breast processes can be reliably accomplished with the apparatus and techniques now at hand, but a comprehensive knowledge and familiarity of the roentgen apearance of the normal breast under all conditions of growth and physiologic activity must be acquired by the roentgenologist before he can ever hope to make real progress in the development of the roentgen method of diagnosis in breast neoplasia.

REFERENCES

1. BIANCHINI, G. Lo studio radiologico della mammella muliebre. *Radiol. med.*, 1935, *22*, 167–172.
2. CHEATLE, G. L., and CUTLER, MAX. Tumours of the Breast. J. B. Lippincott Co., Philadelphia, 1931.
3. DAWSON, E. K. Sweat gland carcinoma of the breast. *Edinburgh M. J.*, 1932, *39*, 409–438.
4. DAWSON, E. K. Histological study of normal mamma in relation to tumour growth. *Edinburgh M. J.*, 1934, *41*, 653–682.
5. DIECKMANN, H. Ueber die Histologie der Brustdrüse bei gestörtem und ungestörtem Menstruationsablauf. *Virchow's Arch. f. path. Anat.*, 1925, *256*, 321–356.
6. DEAVER, J. B., and MCFARLAND, J. The Breast. P. Blakiston's Son & Co., Philadelphia, 1917.
7. ESPAILLAT, A. Contribution à l'étude radiographique du sein normal et pathologique. Thèse de Paris, 1933.
8. FRAY, W. W., and WARREN, S. L. Stereoscopic röntgenography of the breasts as an aid in establishing diagnosis of mastitis and carcinoma. *Ann. Surg.*, 1932, *95*, 425–432.
9. GESCHICKTER, C. F., LEWIS, D., and HARTMAN, C. G. Tumors of breast related to oestrin hormone. *Am. J. Cancer*, 1934, *21*, 828–859.
10. LOCKWOOD, I. H., and STEWART, W. Roentgen study of physiologic and pathologic changes in mammary gland. *J. Am. M. Ass.*, 1932, *99*, 1461–1466.
11. LUSTIG, HILDA. Zur Entwickelungsgeschichte der menschlichen Brustdrüse. *Arch. f. mikr. Anat.*, 1915, *87*, 1. Abt., 38–59.
12. OLIVER, R. L., and MAJOR, R. C. Cyclomastopathy. *Am. J. Cancer*, 1934, *21*, 1–85.

13. Pendergrass, E. P. Discussion of paper by Lockwood and Stewart, ref. 10.
14. Ritvo, M., Butler, P. F., and O'Neil, E. E. Roentgen diagnosis of tumors of breast. *J. Am. M. Ass.*, 1935, *105*, 343–348.
15. Rosenburg, A. Ueber menstruelle, durch das Corpus luteum bedingte Massaveränderungen. *Frankfurt. Ztschr. f. Path.*, 1922, *27*, 466–506.
16. Salomon, A. Beiträge zur Pathologie und Klinik der Mammacarcinome. *Arch. f. klin. Chir.*, 1913, *101*, 573–668.
17. Seabold, P. S. Roentgenographic diagnosis of diseases of the breast. *Surg., Gynec. & Obst.*, 1931, *53*, 461–468.
18. Vogel, W. Die Röntgendarstellung von Mammatumoren. *Arch. f. klin. Chir.*, 1932, *171*, 618–626.

RESOLVING LOBAR PNEUMONIA IN ADULTS SIMULATING TUBERCULOSIS IN THE ROENTGENOGRAM

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OF THE various pathological phases of pneumococcic pneumonia, the stage of resolution from a roentgen viewpoint has probably been least appreciated. Resolution in lobar pneumonia is not only of scientific interest but also of much practical importance. Particularly, in large municipal hospitals where a considerable number of patients enter late in the disease and where the turnover of patients is rapid, the problem of differentiation between some cases of pneumonia and tuberculosis is a vital and not infrequent one. I have had the opportunity of observing a large number of roentgenograms of pneumonia patients. This report includes a group of 10 cases which illustrate especially instructive roentgenological features of resolution of lobar pneumonia in adults. All of them had been incorrectly diagnosed at one time or another as having tuberculosis. The error committed was that of faulty interpretation and evaluation of the chest roentgenograms, particularly of a single film. Limited space will permit data only of representative cases.

CASE REPORTS

CASE I. R. E., colored, female, aged forty-two, admitted to Medical Service on January 10, 1935. *Past history:* Had influenza in 1926. *Family history:* One brother died of pulmonary tuberculosis. *Present illness:* On January 3, 1935, patient developed head cold, chills, fever; on January 4 noticed pain in left chest and cough. *Findings on admission:* Patient obese, moderately dyspneic. Temperature 101.8°F.; pulse 80; respirations 24. The chest showed slight dullness over left upper lobe anteriorly with few crackling râles and rhonchi. *Clinical and roentgenological course:* Temperature came down to normal on January 12, 1935, and remained within normal limits until she left the hospital.

First roentgenogram, January 14, 1935 (Fig. 1) showed haziness plus infiltration in the first and second anterior interspaces (left); physical examination showed an occasional crackling râle below the clavicle. Temperature, pulse and respiration were normal. Patient was discharged on January 18, 1935, and was observed thereafter in the chest clinic. A roentgenogram on February 15, 1935 (Fig. 2)showed that the haziness and infiltration had cleared completely. Chest examination was negative. Temperature, pulse and respirations were normal.

Comment. The first roentgenogram simulated very closely an exudative tuberculous infiltration in the left upper lobe. The fact that the patient was colored and gave a definite contact history seemed to strengthen a diagnosis of tuberculosis. Yet, from the history and subsequent clinical and roentgenologic course which showed rapid and complete disappearance of the infiltration, it is evident that the patient entered the hospital during the resolving stage of a simple lobar pneumonia. The sputum also was negative for tubercle bacilli. A Type VII pneumococcus was present. The patient has had several roentgenograms since February, 1935, and all have been negative.

CASE II. V. T., white, female nurse, aged twenty-seven. *Past history:* Patient had left-sided pneumonia in 1918, followed by a thoracotomy for complicating empyema; influenza in 1932 and pneumonia again in 1933. Since 1932 she has had intermittent episodes of cough with scanty expectoration. *Present illness:* Patient developed typical symptoms of pneumonia on March 16, 1936. Entered Long Island College Hospital, Brooklyn, on March

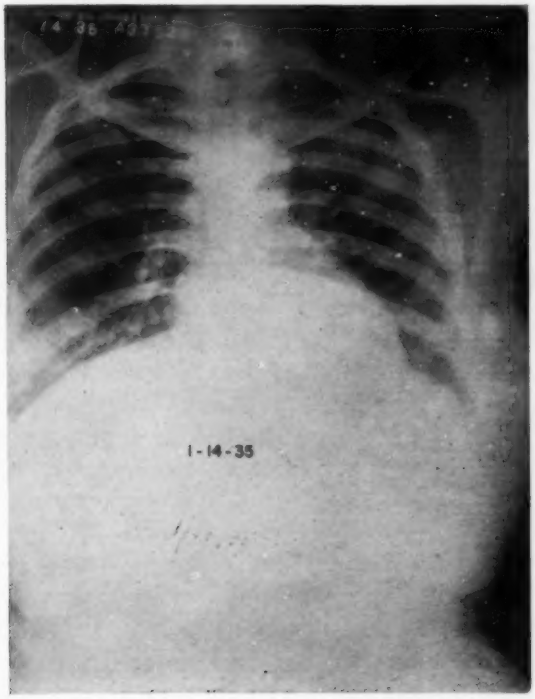

Fig. 1. Case 1. Date of onset of disease, January 3, 1935.

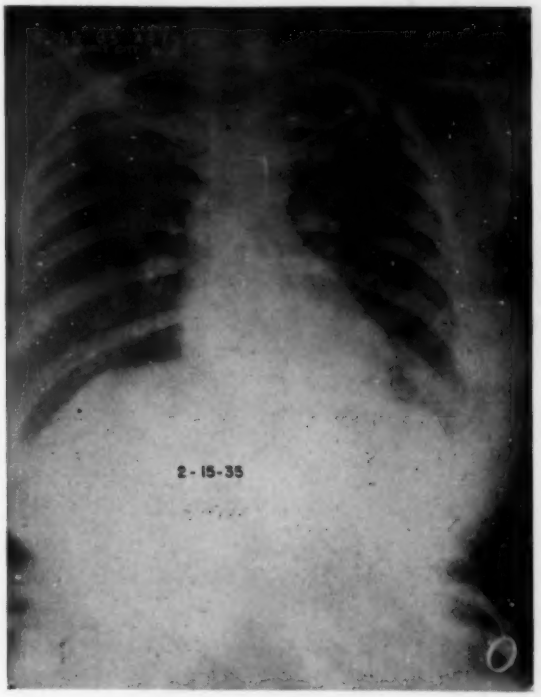

Fig. 2. Case 1.

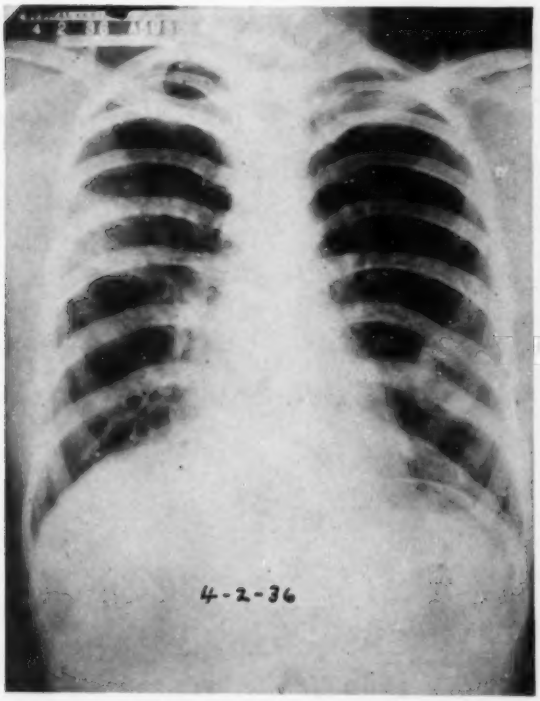

Fig. 3. Case II. Date of onset of disease, March 16, 1936.

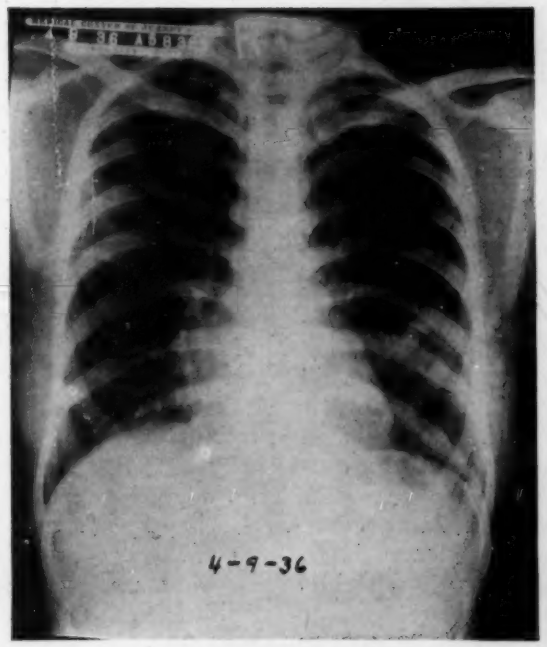

Fig. 4. Case II.

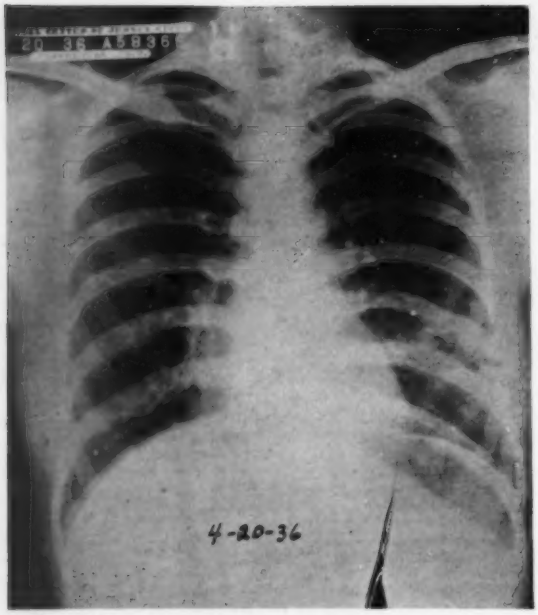

Fig. 5. Case II.

18, 1936. A diagnosis of right upper lobe pneumonia was made. Type II pneumococcus was recovered from the sputum and four serum injections were given. She was discharged March 28, 1936. *Clinical and roentgenological course:* Cough and expectoration with low grade fever ranging from 100° to 100.6°F. continued after leaving the hospital. The patient, a graduate of the Jersey City Medical Center Training School, was referred to our chest clinic April 2, 1936. A roentgenogram on April 2, 1936 (Fig. 3) showed a dense band of infiltration in the peripheral portions of the second and third anterior interspaces (right); very slight infiltration in the first anterior interspace (right); defect in the fourth left anterior rib due to old thoracotomy. Physical examination revealed slight dullness from apex to the fourth anterior rib with bronchovesicular breathing and coarse râles. A roentgenogram on April 9, 1936 (Fig. 4) showed considerable clearing with very slight residual infiltration in the periphery of the second and third anterior interspaces; coarse râles still persisted; temperature was normal. The infiltration completely cleared in the film of April 20, 1936 (Fig. 5); physical signs were unchanged; temperature, pulse and respirations, normal. A bronchogram on June 6, 1936 (Fig. 6) revealed definite cylindrical bronchiectasis in the right middle lobe; cough with expectoration of 2 to 3 drachms of non-foul mucopurulent sputum still existed; some coarse râles between the second and fourth interspaces (right) were present. Temperature, pulse and respiration, normal.

Comment. An exudative type of pulmonary tuberculosis had been suspected elsewhere almost solely on the basis of the single roentgenogram of April 2, 1936. Sputum was negative for tubercle bacilli; Mantoux test, positive. Frequent roentgenograms showed rather rapid clearing of the infiltration. Obviously, the patient entered our clinic during the resolving phase of her pneumonia. The persistence of cough and expectoration and râles in the chest together with the past history of repeated respiratory infections indicated an underlying predominantly dry type of bronchiectasis. This was confirmed by the bronchogram.

Case III. M. G., white, female, aged fifty-nine, admitted March 4, 1935. *Past history:* Negative. *Present illness:* On March 7, 1935, patient developed pain in right hypochondrium

Fig. 6. Case II.

which radiated to the right scapula; slight cough and vomiting were also present for several days before admission. *Findings on admission:* Patient was acutely ill; temperature 99.2°F., pulse 94, respirations 24. The chest showed dullness, bronchial breathing and coarse râles present over the right upper lobe posteriorly. *Abdomen:* Definite tenderness elicited in the right upper quadrant. *Clinical and roentgenological course:* The temperature ranged between 99°–103.4°F. until March 20, 1935, and then oscillated between 98° and 99.8°F.

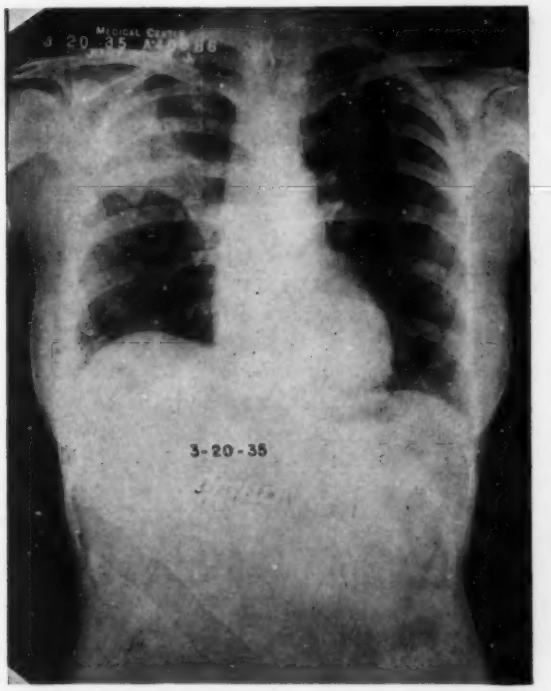

Fig. 7. Case III. Date of onset of disease, March 7, 1935 (?).

until discharge on April 7, 1935. The first roentgenogram of March 20, 1935 (Fig. 7) showed a fairly homogeneous density demarcated by the interlobar fissure at the level of the third anterior rib and extending from the right hilum into the second anterior interspace in a fan-shaped manner. This density feathered off into a less dense infiltrate in the first anterior interspace; in the mid-zone of the second interspace was a small area of suggestive "high light"; slight infiltration at extreme left apex also; the chest signs were as noted above; temperature 98–99.2°F., pulse 88–94, respirations 22. The chest roentgenogram of April 3, 1935 (Fig. 8) revealed considerable clearing of

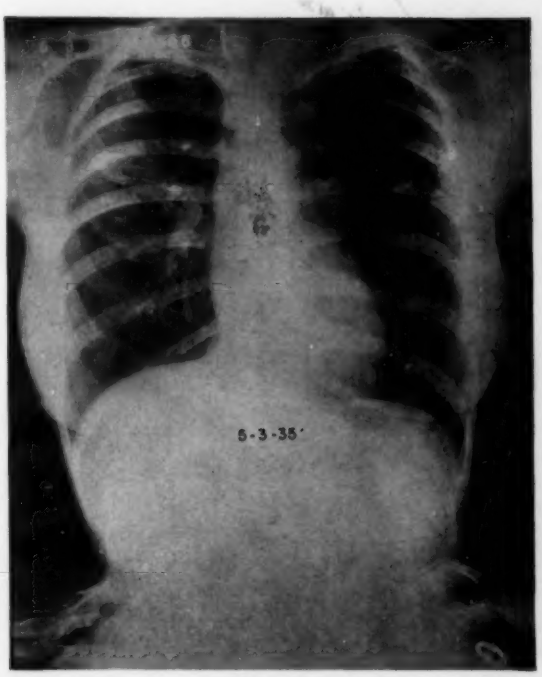

Fig. 8. Case III.

the density but an annular shadow (1.5×2.0 cm. in diameter) situated just below the second anterior rib (right) was much more conspicuous. General condition good; temperature

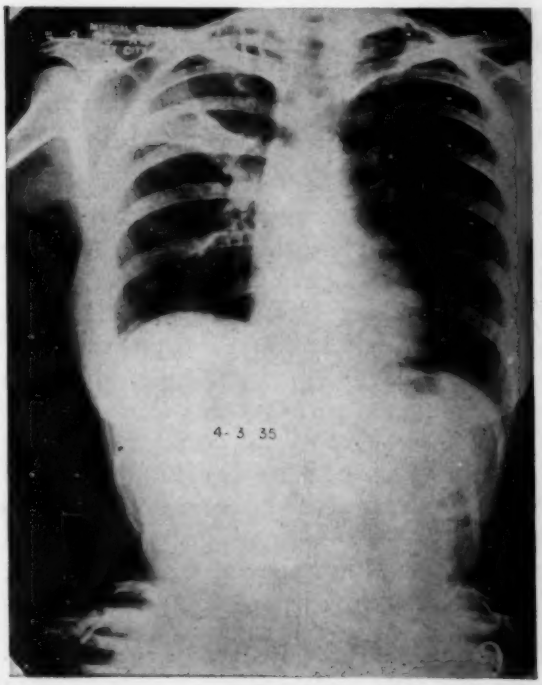

Fig. 9. Case III.

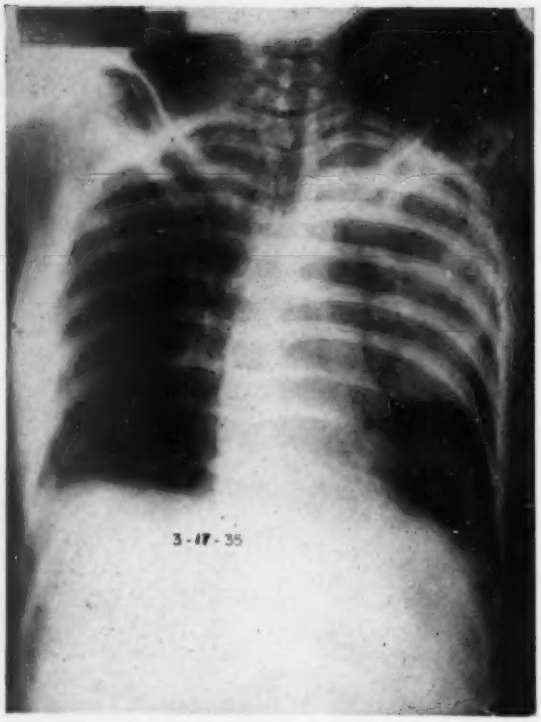

Fig. 10. Case iv. Date of onset of disease, March 7, 1935.

98.8°–99.8°F., pulse 70–84, respirations 24–26. The annular shadow entirely cleared in the subsequent film of May 3, 1935 (Fig. 9); pulmonary markings were somewhat increased; thickened interlobar fissure was seen crossing the second anterior rib. Roentgenograms of the gallbladder, incidentally, showed two calculi.

Comment. A roentgen diagnosis of pulmonary tuberculosis was made on the basis of a single film, for the "ring" shadow was interpreted as a cavity. Subsequent course with clearing of physical signs, return to normal temperature and disappearance of abnormal densities on the roentgenogram —all indicated a diagnosis of lobar pneumonia. Type iv pneumococcus was present in the sputum which was also negative on three occasions for tubercle bacilli. I believe that the first roentgenogram taken thirteen days after the onset of the present illness showed evidence of resolution.

Case iv. M. B., white female, aged twenty-nine. *Past history:* Patient had pertussis at nine years of age and pneumonia at ten. Cough with expectoration continued and she spent two years at a tuberculosis sanatorium between the ages of twelve and fourteen. She returned to the sanatorium for a period of six months at the age of eighteen. *Present illness:* On March 7, 1935, patient developed increased cough, chills, fever. She was five months pregnant at the time and entered the Margaret Hague Maternity Hospital on March 10, 1935. *Findings on admission:* Patient was very toxic with a temperature of 103.6°F. The chest showed dullness in left upper lung posteriorly, with increased fremitus, bronchial breathing and crepitant râles, similar râles were also present posteriorly over the lower lobe; sonorous râles were heard in left lung anteriorly and posteriorly. *Clinical and roentgenological course:* A portable roentgenogram of March 11, 1935 (Fig. 10) showed a rather solid density on the left side extending from the apex to the third anterior interspace; generalized increase of linear markings with strands of fibrosis noted; calcified focus in first anterior interspace and productive infiltrate below second anterior rib (right) with flattening of diaphragm also seen. Temperature 100°–103.6°F., pulse 120 to 140, respirations 28. The second roentgenogram of March 27, 1935 (Fig. 11) showed mottled

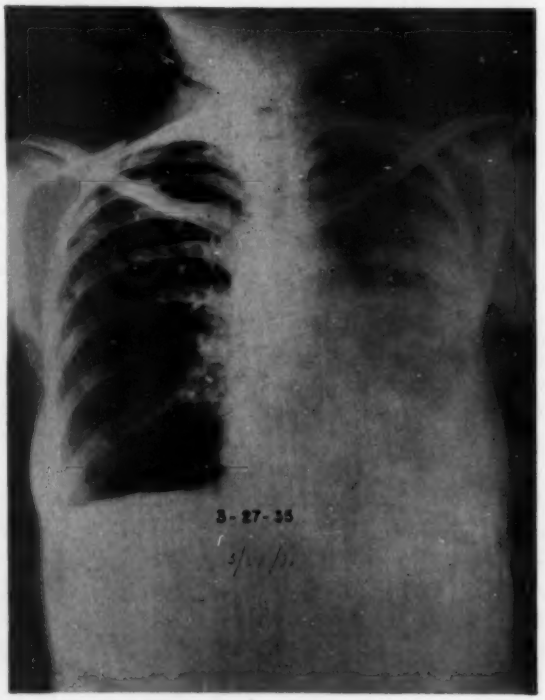

Fig. 11. Case iv.

infiltrative involvement throughout greater portion of left lung with haziness in the lower two-thirds; physical examination disclosed some clearing of abnormal signs several days previously. Temperature 99.2°F., pulse 110. On April 2, the patient was delivered of a six months' stillbirth. A diagnosis of pulmonary tuberculosis was strongly entertained and she was transferred to the Tuberculosis Service April 10, 1935, but signed release the same day. However, she was induced to report for follow-

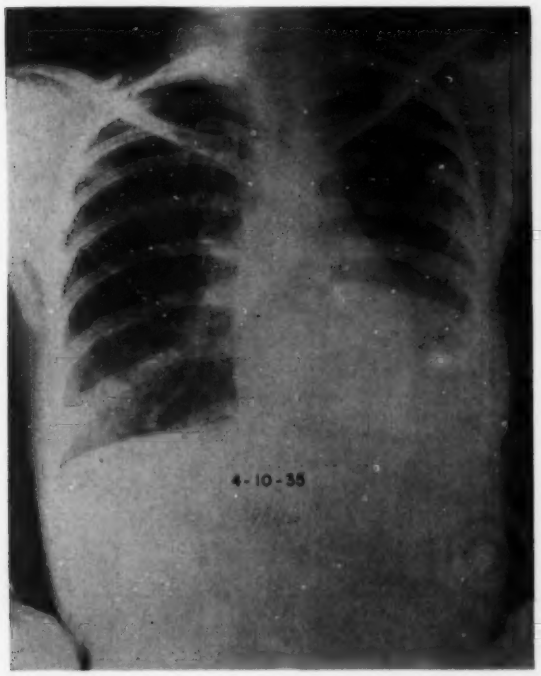

Fig. 12. Case IV.

up examinations. Cough and expectoration of about ½ ounce of mucopurulent sputum persisted. Another chest film of April 10, 1935 (Fig. 12) revealed considerable resorption of the infiltrate with a haziness at the left base. Temperature 99.2°F. In the roentgenogram of April 26, 1935 (Fig. 13) almost complete clearing of the infiltration in the upper half of left lung field was noted. Dullness over the left upper lobe with bronchovesicular breathing and coarse râles still present. Similar râles were heard at base posteriorly. The patient entered the Medical Service June 18, 1935 because of a vaginal hemorrhage, increased cough and fever. She was again transferred to the Tuberculosis Ward on July 3, 1935. The roentgenogram of July 5, 1935 (Fig. 14) showed distortion

Fig. 13. Case IV.

of the left-sided cardiac contour with a loss of the normal convexity, most likely due to pleural pericardial adhesions; a "shrunken" lower left lobe was present; increased markings and strands of fibrosis throughout the left lung.

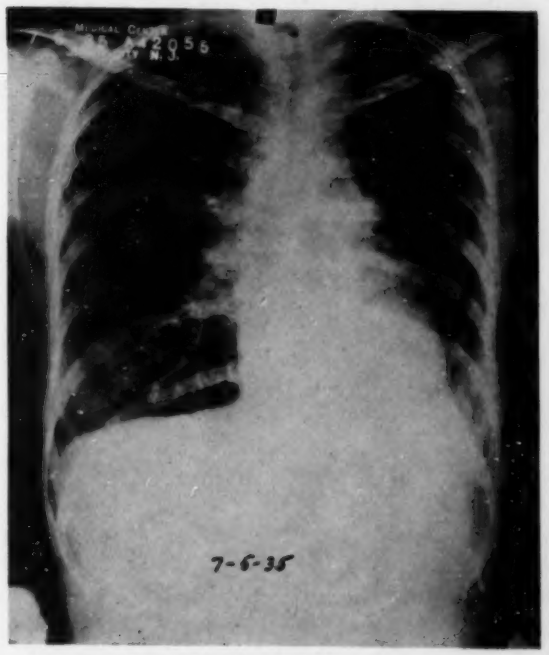

Fig. 14. Case IV.

Fig. 15. Case iv.

The right lung was unchanged. Physical examination of the right lung revealed dullness in supraclavicular fossa and posteriorly over upper lobe and coarse râles at base posteriorly. The left lung presented dullness throughout, more marked below inferior angle of scapula; bronchial breathing from fifth vertebral spine to base with numerous moist râles; dullness anteriorly to fourth anterior rib with fewer coarse râles. Temperature at this time was 98°–99.2°F. and stayed approximately in this range until she left the hospital on September 9, 1935. A bronchogram of July 17, 1935 (Fig. 15) showed marked saccular bronchiectasis in the left lower lobe. Subsequent bronchograms also revealed cylindrical and saccular bronchiestasis in the right middle and right lower lobes but none in that part of the left upper lobe which was mapped out by lipiodol.

Comment. Pulmonary tuberculosis was strongly suspected at first because of the patient's previous sojourn in a tuberculosis sanatorium, the long history of pulmonary complaints and the roentgenographic findings of March 27 and April 11, 1935. The Mantoux test was positive. However, a positive sputum for tubercle bacilli had never been obtained on this patient. During her last illness a Type iv pneumococcus was recovered; the sputum was also injected into a guinea pig which showed no evidence of tuberculosis on necropsy. The history of respiratory illness in childhood was very significant. The final diagnosis was that of a slowly resolving left upper lobe lobar pneumonia with extension to the lower lobe, superimposed upon a low grade chronic pneumonitis with bronchiectasis.

Case v. E. P., white female, aged thirty-eight, a mental defective, was admitted to the Psychiatric Service March 2, 1934 and transferred to the Tuberculosis Service March 6, 1934. *Past history:* Patient had pneumonia and empyema (right) in childhood with subsequent thoracotomy. Frequent attacks of bronchitis had occurred since then. *Present illness:* About two weeks before admission, she developed pain in right anterior chest, increased cough with 2 ounces of mucopurulent sputum daily; had lost some weight. *Findings on admission:* Patient fairly well nourished, moderately dyspneic. Temperature 97.8°F.; pulse 108; respirations 26. The right lung showed dullness, bronchial to bronchovesicular breathing

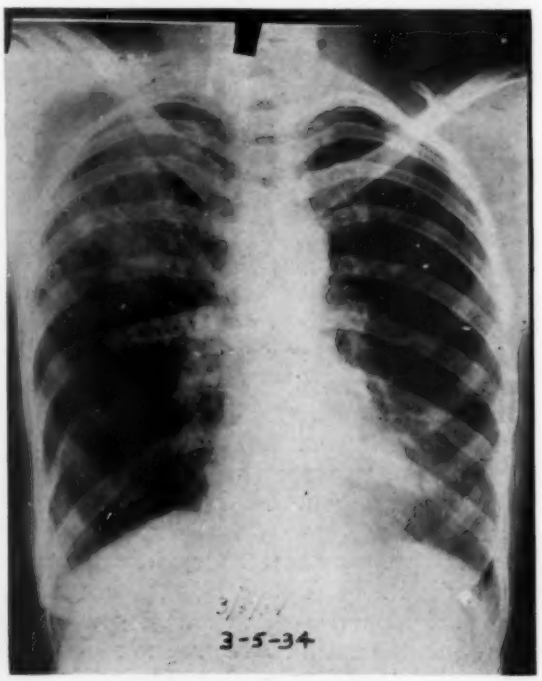

Fig. 16. Case v.

with coarse râles over upper lobe anteriorly and posteriorly. Few crackling râles were noted over left apex posteriorly. Heart: Negative. *Clinical and roentgenological course:* Temperature ranged between 97.8°–99.8°F. from March 2, 1934, to March 28, 1934. From March 29, 1934, to April 3, 1934, temperature varied from 97.6°–102.6°F. and thereafter was within practically normal limits. The first roentgenogram of March 5, 1934 (Fig. 16) revealed patchy infiltration on the right from the apex to the third anterior rib with small suggestive honeycombed areas; thickened interlobar fissure in third interspace; large calcified node at hilum; irregularity of diaphragm; defect of eighth rib due to old thoracotomy. In the left lung field was an increase in linear markings with few strands of fibrosis at extreme apex and in first anterior interspace. Blood count on March 3, 1934, showed total leukocyte count of 8,700 cells with 76 per cent polymorphonuclears. Wassermann reaction was four plus and antiluetic therapy was instituted. The second roentgenogram of March 22, 1934 (Fig. 17) showed definite decrease in the right-sided infiltration. Temperature was 100.8°F. In the third chest film of May 9, 1934 (Fig. 18), complete disappearance of the infiltration was apparent; temperature was normal. Follow-up examinations were negative for tuberculosis. For various reasons, the patient was kept under observation at the Medical Center until January, 1935, and then transferred to a county institution.

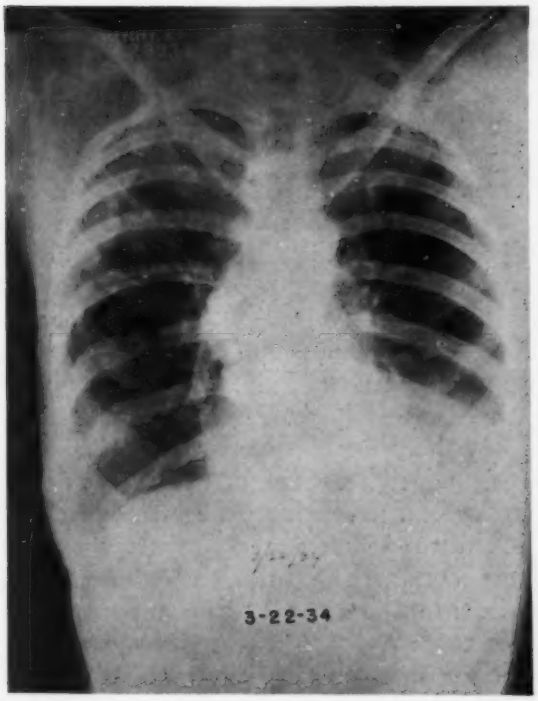

Fig. 17. Case v.

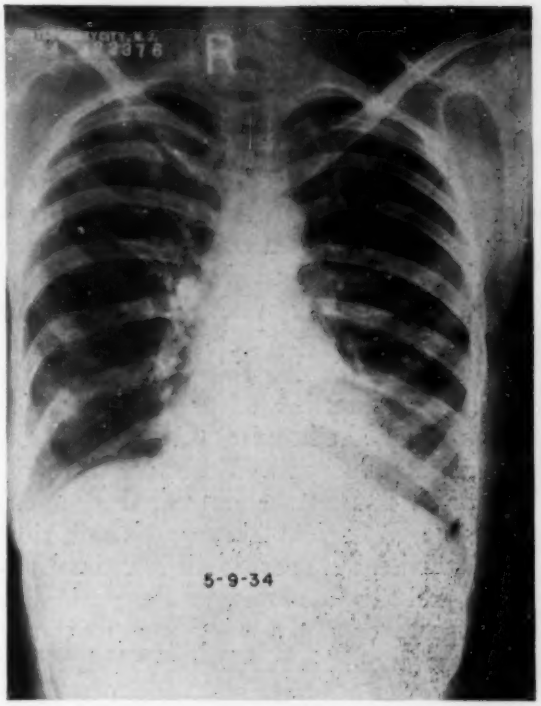

Fig. 18. Case v.

Comment. This case was diagnosed, at the outset, as one of pulmonary tuberculosis especially on the basis of the type and distribution of the mottling visualized on the first film. The presence of the large calcified hilar gland seemed added justification for this diagnosis. However, serial roentgenograms revealed too rapid clearing for a possible tuberculous lesion of this extent without more definite roentgenographic residue. Twenty-three sputum examinations were negative for tubercle bacilli. A Type iv pneumococcus was present. I believe the patient entered the hospital during the resolving stage of a lobar or lobular pneumonia. Roentgen differentiation between the two, during resolution, may be almost impossible at times. Furthermore, contrary to the old teaching, bronchopneumonia is often unilateral and

may occur in an upper lobe which also may make its differentiation from tuberculosis difficult.

DISCUSSION

Resolution of the exudate in lobar pneumonia with the return to normal pulmonary tissue is truly a remarkable process. Yet the literature on this phase of the infection is relatively small and incomplete because: (1) many patients are not followed closely enough during this stage and (2) a considerable number of patients are discharged from the hospital before the lesion has entirely cleared and are lost sight of.

In order to best understand the clinical and roentgenological features of resolution together with our specific differential diagnostic problem, a review of the pathological aspects of a resolving pneumococcic lobar pneumonia is pertinent at this time.

A. Pathology of a Normally Resolving Unilobar Pneumonia. MacCallum[9] states that the stage of resolution is seldom seen at autopsy unless the individual dies of some intercurrent infection such as meningitis, etc. However, the excellent and detailed work of experimentally induced pneumococcic pneumonia in monkeys (recorded by Blake and Cecil[2]), and in dogs (reported by Robertson, Coggeshall and Terrell,[12] among others) is available to us and helps fill in some gaps. The process of resolution occurring in these animals is almost exactly as in humans. Recently also, *intra vitam* punch biopsy studies in individuals recovering from lobar pneumonia have been undertaken which supplement our post-mortem and experimental data (Curphey[4]).

Grossly, the affected lobe is somewhat smaller than normal, softer and more friable than in the stage of gray hepatization and usually has a greenish-yellow color. Microscopically, the criterion for the onset of resolution is what Robertson *et al.*[12] characterize as the "macrophage reaction." The alveolar walls become thickened by active proliferation and swelling of the alveolar epithelium. Large macrophages soon make their appearance but the origin of these cells is still a controversial point. Some believe that they are desquamated alveolar epithelial cells, others that they are derived from the blood. These are the active phagocytic cells, digesting polymorphonuclear leukocytes, red blood cells, fibrin and pneumococci. In addition, pigment-bearing macrophages are found. The actual dissolution, however, is accomplished by proteolytic ferments arising from the disintegrated leukocytes. Finally the liquefied débris is removed, (*a*) by absorption and elimination through the kidneys; (*b*) partly by cough and expectoration, and (*c*) by absorption through the lymphatics. The lymph vessels are often inflamed, forming yellowish streaks through the lung and lead ultimately to the hilar glands which become enlarged. Peribronchial and perivascular edema with exudation and some engorgement of the capillaries are also present but recede rapidly. As these changes continue, the lung becomes aerated, epithelial repair takes place and under ordinary conditions, there is a rapid restoration to normal.

B. Roentgen Appearance of a Normally Resolving Unilobar Pneumonia. How are these pathological changes reflected on the roentgenogram? The criterion for beginning resolution, roentgenographically, is a decrease in the extent or intensity of the opaque density characteristic of the stage of hepatization. The first site of resolution is variable. It may manifest itself in the peripheral or hilar zones or in the center of the dense shadow. A diffuse haziness is frequently an early finding which may clear rather uniformly or rapidly or more commonly becomes broken up into smaller areas of lighter infiltration which are patchy in distribution. Roentgenograms of the chest repeated several days apart often show complete clearing at some point, while in others infiltration may persist. This corresponds to the pathological evolution of the process. Many times there are seen extending from the hilum in all directions, linear streaks which cross one an-

other and also intersect larger areas of infiltration, giving the appearance of a reticulum. It is difficult to say whether these lines are due predominantly to vascular or lymphatic dilatation. I am inclined to believe with Impallomeni[7] who takes the latter view and states that absorption of the exudate comes about mostly via the lymphatics which become distended and are revealed by the roentgenogram. Hyperemia is greatly reduced in the resolving stage. He emphasizes the roentgenological similarity between this phase of pneumonia and endolymphatic pulmonary carcinomatosis.

In a lung field so disorganized, the presence of annular shadows is not infrequent. These may be due simply to a ring-like configuration of markings or actually to distended air sacs which Robertson *et al.* found in the dog. The infiltration disappears, the lacunar shadow or shadows become thinned out and clear and the pulmonary markings are the last to take on their normal appearance.

As previously noted, glandular enlargement at the hilum is present but I have only rarely been able to dissociate with certainty its roentgen visualization from the parenchymal process. However, only posteroanterior films were taken. The same discrepancy between roentgenological and pathological findings is also common in chronic pulmonary disease.

Displacement of the mediastinal structures, particularly the trachea, to the affected side is very occasionally seen in the resolving stage. Graeser, Wu and Robertson,[6] on the basis of the experimental work of Van Allen and Wu,[13] believe that this is a compensatory shift which is due to decreased expansibility and reduced volume of the lung during this phase.

The time required for complete resolution with return to a normal roentgenogram varies. A maximum period of three weeks after the date of onset of the disease is a good rule for the normal complete resolution of a unilobar pneumococcic pneumonia in adults.

C. Roentgen Differentiation between Resolving Lobar Pneumonia and Tuberculosis. In these days, when there is a tendency to look upon roentgenograms of the chest as the final ante-mortem arbiter in the diagnosis of pulmonary disease, there is present an inevitable pitfall in the differentiation between these infections. I wish, therefore, to emphasize the following points in our roentgenological diagnostic problem:

(1) The roentgenogram in the resolving stage of lobar pneumonia may pattern itself very closely after the manner of the infiltrative or cavernous types of tuberculosis as may be inferred from the preceding description and as the case histories well illustrate. The difficulty of the problem is increased when the lesion is confined to the preferred site of tuberculosis, namely, the upper half of the lung field. I have already alluded to the pathological basis for the thin-walled annular shadows. These are transient but are often mistaken for pulmonary cavities. We must remember that in reading a roentgenogram we are simply interpreting various densities which are not necessarily pathognomonic for any specific pulmonary disease. We must avoid a common source of error and not make an etiological diagnosis on the basis of a single roentgenogram.

(2) One of the most important differential points hinges on the *time interval* for resolution of the pulmonary process. Resolution in pneumonia may first occur at the time of the crisis but may also antedate or follow it. While there is considerable variation, clearing of the infiltrative and ring shadows usually takes place in seven to fourteen days after the crisis in uncomplicated cases. Another significant point is the *absence of a residue* of the pulmonary infection on roentgen examination as resolution is completed. Pulmonary tuberculosis, on the other hand, resolves much more slowly over periods of weeks and months. Amberson,[1] in an illuminating article, writes:

> The slow rate of resolution of tuberculous lesions is characteristic. Starting, as it does, in

their peripheral parts, it may be seen roentgenographically to proceed toward the center with impressive rapidity for several weeks. Thenceforth, the change is usually slow though it may continue for months; and finally it stops at the barrier of the unresolvable caseous center or centers. . . . Resolution affects non-caseous inflammatory exudate, but does not influence caseous deposits of significant size.

These latter lesions, on healing, leave behind on the roentgenogram a residue in the form of strands or nodules of fibrosis or calcium deposits. However, Amberson notes that "Small caseous lesions may persist even though invisible on the X-ray film." The difference between the two infections in these respects is the difference fundamentally between a destructive and a non-destructive process in the lung. Ordinarily, pneumococcic lobar pneumonia is really a superficial infection of the lung, producing no necrosis of alveolar or bronchial walls or interstitial tissue. This accounts for the rapid and complete return to normal.

I am of the belief that some cases labelled as acute benign exudative tuberculosis are truly cases of lobar pneumonia or bronchopneumonia seen by the physician in the resolving period. It is absolutely imperative that the history and entire clinical course be fully evaluated. The tuberculin test also finds its place here. While a positive reaction has no specific diagnostic value in the adult as a differential point, a negative reaction to 1.0 mg. of old tuberculin in a case suspected of pulmonary tuberculosis should make one strongly believe that the case has been misdiagnosed, provided none of the conditions are present which would depress the allergic response. Repeated sputum examinations for tubercle bacilli are also of paramount importance. If, in addition, we rely not on a single roentgenogram but serial roentgenograms of the chest taken at short intervals in order to compare the rapidity and residuum of resolution, the diagnosis between resolving lobar pneumonia and pulmonary tuberculosis will almost always be quite evident.

(3) Thus far, we have been concerned only with a normally resolving lobar pneumonia. The difficulty in differential diagnosis is more manifest in the atypical cases of *delayed resolution* particularly when present in the upper lobe. We are not concerned, here, with the sequels of empyema and abscess formation which, strictly speaking, are not true examples of prolonged resolution. While the agents in its production are not yet completely understood, I shall refer briefly to pertinent observations mentioned in the literature. These points should be considered in the problem of differential diagnosis.

Factors in Delayed Resolution. (*a*) Sex—delayed resolution occurs more frequently in males than in females. (*b*) Race—there is a relatively higher incidence among the colored race than among the white. (*c*) Location of lesion—the right lower lobe is involved more often than any other lobe. (*d*) Age—advanced age does not have any influence on resolution. These were the conclusions of McCrae[10] from his study of 40 cases of delayed resolution. Davies *et al.*[5] believe, however, that as a rule resolution is slower in the older age group. Others are of the opinion, too, that delayed resolution is more common in upper lobe involvement. (*e*) Unilobar lesions resolve more quickly than multilobar lesions. (*f*) Administration of serum—this apparently does not hasten resolution according to Davies and his coworkers. Dr. R. L. Cecil,[3] on this same subject, writes, "I am not sure if anti-pneumococcus serum hastens resolution of the pulmonary process in pneumococcus pneumonia, unless it is given extremely early in the disease. It would seem to me, resolution is more or less a mechanical process, that while serum destroys the pneumococcus and its toxin, a certain amount of time would always be required for resolution of the exudate. When serum is given very early, I am convinced that it checks the spread of consolidation." (*g*)

Delayed resolution occurs about as frequently in syphilitics as in non-syphilitics. So concluded Weinstein and Goodman[14] who reviewed 509 cases of acute lobar pneumonia from the Johns Hopkins, Vanderbilt University and Baltimore City Hospitals. (*h*) Resolution will very often be delayed if lobar or bronchopneumonia is superimposed upon a previously damaged lung (as in silicosis, bronchiectasis, tuberculosis, etc.) or when it occurs as an acute exacerbation of a chronic suppurative pneumonitis. Davies *et al.*, in a study of 119 cases of pneumococcic pneumonia, also noted that the presence of calcified hilar glands frequently meant delay in resolution. Lord[8] mentions these local factors: (1) impaired circulation through the involved area; (2) an excess of serum or a deficit of proteolytic ferments may delay resolution; (3) the local hydrogen ion concentration—increasing acidity will hasten liquefaction of the exudate.

McCrae defined delayed resolution as a delay in the process of liquefaction or absorption and removal of the exudate. He found an incidence of 3.7 per cent in 805 pneumonia cases at the Johns Hopkins Hospital. Only cases which showed a persistence of signs for a period of longer than three weeks were included. Musser and Norris[11] found an incidence of 4.1 per cent in 2,548 collected cases. I have been impressed by the fact that the roentgenogram at times reveals a residual lesion when abnormal symptoms or signs are no longer present. Because of their apparent well being, these patients are discharged from the hospital. If pneumonia patients were followed more closely by means of frequent roentgenographic examinations, this discrepancy would be recognized more readily and the incidence of delayed resolution, I believe, would be higher than that reported by older authors who relied on the persistence of symptoms and signs as their criterion. The check-up physical and roentgen examinations that we give our tuberculosis patients should be extended to pneumonia patients if we wish to clarify our ideas on delayed resolution and the relationship of pneumonia to pulmonary fibrosis and bronchiectasis. These examinations should also guide us in determining the duration of the convalescent period. No patient should be allowed to resume his occupation before there has been complete clearing of the acute process on the roentgenogram.

Cases with protracted resolution may bring anxiety to the physician, tax his patience and cause a premature incorrect diagnosis of tuberculosis. However, if one correlates carefully the history, clinical course and roentgenological features which have been previously mentioned, the diagnosis between lobar pneumonia and tuberculosis need not be such a perplexing problem.

SUMMARY

Differentiation between resolving lobar pneumonia and pulmonary tuberculosis, especially among patients in large municipal institutions, is not an uncommon problem, and is of vital concern to the individual. The basis of this report is a group of 10 cases of resolving pneumonia in adults who were erroneously suspected of having tuberculosis. Five of these cases are briefly presented with serial roentgenographic studies. For roentgen differentiation between the two diseases, the value of such serial roentgenograms taken at short intervals is particularly emphasized.

The pathologic and roentgen appearances of a normally resolving lobar pneumonia are reviewed. Potential pitfalls and differential points in the roentgenological diagnosis are stressed. Closer roentgen observation of pneumonia patients during the resolving stage and follow-up examinations after leaving the hospital are recommended.

I am indebted to Dr. B. S. Pollak for some of the cases reported which were admitted to his Tuberculosis Service at the Jersey City Medical Center. For the others, I wish to acknowledge the courtesy extended to me by Drs. Street and Immanual Pyle,

attending physicians on the Medical Services and by Dr. Emmet Connell, who kindly permitted me to follow and report these cases.

REFERENCES

1. AMBERSON, J. B., JR. Process of resolution in pulmonary tuberculosis. *Am. Rev. Tuberc.*, 1936, *33*, 269–301.
2. BLAKE, F. G., and CECIL, R. L. Studies on experimental pneumonia; pathology and pathogenesis of pneumococcus lobar pneumonia in monkeys. *J. Exper. Med.*, 1920, *31*, 445.
3. CECIL, R. L. Personal communication.
4. CURPHEY, T. J. Preliminary report on *intra vitam* biopsy studies of pathogenesis of pneumococcus lobar penumonia. *Am. J. Path.*, 1935, *11*, 861.
5. DAVIES, D. T., GRAHAM HODGSON, H., and WHITBY, L. E. H. Study of pneumococcal pneumonia. *Lancet*, 1935, *1*, 919.
6. GRAESER, J. B., WU, C., and ROBERTSON, O. H. Physical signs and roentgenographic findings in lobar pneumonia in adults. *Arch. Int. Med.*, 1934, *53*, 249–268.
7. IMPALLOMENI, R. L'indagine radiologica nella risoluzione della polmonite lobare. *Radiol. med.* 1934, *21*, 404–423.
8. LORD, F. T. Bearing of the evolution of pneumonia exudate on local chemical factors influencing recovery and resolution in lobar pneumonia. *Tr. Ass. Am. Physicians*, 1921, *36*, 61.
9. MACCALLUM, W. G. A Text-Book of Pathology. W. B. Saunders Co., Philadelphia.
10. MCCRAE, T. Delayed resolution in lobar pneumonia. *Johns Hopkins Hosp. Rep.*, 1910, *15*, 277–305.
11. Osler's Modern Medicine, 1925, *1*, 184.
12. ROBERTSON, O. H., COGGESHALL, L. T., and TERRELL, E. E. Experimental pneumococcus lobar pneumonia in the dog; pathogenesis. *J. Clin. Investigation*, 1933, *12*, 467–493.
13. VAN ALLEN, C. M., and WU, C. Increased elastic tension of lung in experimental pneumonia. *J. Clin. Investigation*, 1932, *11*, 589–605.
14. WEINSTEIN, A., and GOODMAN, M. J. Delayed resolution in lobar pneumonia and its relation to preëxisting syphilis. *Am. J. M. Sc.*, 1934, *188*, 716–721.

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ROENTGENOLOGICAL MANIFESTATIONS IN BONE SYPHILIS

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THE antiquity of syphilis has been proved by many investigators, who have based their conclusions upon the known bone changes in this disease. The controversy appears to be between those advocating the Columbian[2,3] and others supporting the pre-Columbian[23,24] origin of lues, each side making use of various bones unearthed in archeological researches. However, the interesting point which is made use of is the definite pathological alteration in the normal osseous structures. Williams,[24] in a recent supplementary report, gives credit to Dr. Joseph Jones,[6] of New Orleans and Dean of the Medical College of Louisiana, which later became Tulane University, for first bringing to light the question of the antiquity of syphilis by the study of ancient bones.

That there is a distinct alteration in the structure of bones due to systemic infection with the *Treponema pallidum* there can be no doubt, and a recognition of these changes, which in most instances are rather characteristic, is a distinct aid in the diagnosis of a hitherto hidden or latent syphilitic background. The medical profession is prone to place its faith more or less entirely in the various complement-fixation and precipitation reactions of the patient's blood serum, and in instances in which, for unexplained reasons, this reaction is negative, the search for other evidences of the disease is neglected. By a careful study of the osseous system, both in the newborn, and in adults with the possibility of acquired syphilis, the striking bone changes will often give at least a suspicion of the presence of the disease. Since the manifestations of syphilis tend to mimic practically every other known disorder, it is true that these lesions may be mistaken for other conditions, but with some knowledge of the changes peculiar to syphilis from a roentgenological examination of the bones and joints, the presence or absence of such a background will be a valuable aid in establishing the possibility of the suspected, or unsuspected infection.

The literature contains many interesting articles of the unexpected prevalence of congenital syphilis. This frequency in many instances is predicated upon the typical bone and joint changes, especially where the skin and visceral manifestations are absent, or where they may be slight. There is the interesting finding of Shipley *et al.*[17] that 15 per cent of fetuses, which were considered normal in the Carnegie Institute of Embryology, presented unmistakable syphilitic bone lesions. The excellent work of McLean[10,11,12,13] also points to the relatively large percentage of congenital syphilis prevalent, which may be identified, or at least suspected by the roentgen examination of the bones and joints.

Until 1870, syphilitic osteochondritis was considered to be extremely rare. Wegner,[22] in his carefully prepared article, however, showed that this condition was typically luetic. The pseudoparalysis of Parrot was described in 1869. The frequency of bone manifestations in congenital syphilis was first called to the attention of the medical world by Parrot,[15] Waldeyer and Köbner[21] in 1872, and Taylor[18] in 1874, and later, Holt,[5] in 1911, commented on the large numbers of infants with the recognizable osseous lesions of congenital lues.

With the advent of the growing efficiency in the field of roentgenology, and the early recognition of these bone changes, this investigation received its greatest stimulus. Lovett and Wolbach,[9] Turnbull,[20] Pendergrass and Bromer,[16] Knaggs,[7] O'Reilly,[14] and McLean,[10,11,12,13] brought forth a new

knowledge in the pathology of these bone manifestations, and in their roentgenological significance. These writers are but a few who have contributed to our knowledge of congenital bone and joint syphilis.

BONE AND JOINT CHANGES IN CONGENITAL SYPHILIS

Classification

1. Epiphysitis
2. Periostitis
3. Epiphyseal separation
4. Circumscribed rarefying osteitis of long and flat bones.
5. Dactylitis—rare
6. True bowing of tibia in lower two-thirds
7. Pathological fractures—rare
8. Osteomyelitis
9. Osteochondritis
10. Parrot's nodes on the flat bones

The earliest manifestations of congenital syphilis may be noted in the first months of life, although it may not be evident until puberty or later. The long bones are chiefly affected, particularly the tibia and the shafts more than the epiphyses. The juxta-epiphyseal region is widened and the epiphyseal line is indistinct, there being a haziness about this region.

Another early manifestation of syphilis is periostitis. There is a distinct thickening of the periosteum, particularly on the shaft of the tibia. This periostitis is always to be considered a distinct diagnostic feature.

The epiphyseal lesion may be readily distinguished from early rickets. In rickets the epiphyseal ends of the bones are distinctly saucer shaped, and the epiphyseal line is widened and extremely irregular with evidence of decreased bone density on either side of it. Scurvy presents a different periosteal manifestation, there being an elevation of the periosteum due to a subperiosteal hemorrhage. In the earlier stage the recognition of this is difficult, but later, with the beginning calcification of this hemorrhagic mass, it becomes evident. There is also a distinct area of calcification behind the epiphyseal line together with a characteristic ring about the margin of the smaller epiphyses.

In the early stages a multiplicity of bone lesions is common. These lesions, however, have no permanency and they disappear within a comparatively short time whether the patient has been treated or not. This applies chiefly to the characteristic periosteal proliferation.

Along with the juxta-epiphyseal haziness and periosteal proliferation one may find simultaneously a distinctive epiphysitis. This condition, if untreated, will persist and produce deformities later. Other changes commonly seen are destructive osteitis which is found at the end of the long bones. The pseudo-paralysis of Parrot is produced by this involvement. It consists in a sudden loss of use of the extremities with swelling of the joint and pain on motion. These symptoms usually disappear in several weeks even in the absence of treatment. Occasionally, however, the destruction about the epiphyseal line is sufficiently great to produce an epiphyseal separation. It is seldom that a single joint is so involved. Another peculiar appearance common to this condition is the circumscribed areas just beyond the epiphyseal line which extend into the bone itself. It should be remembered,[1] however, that luetic infections nearly always produce new bone.

Involvement of the phalanges and metacarpals of the upper extremities is not very common, but when it occurs it persists for a long time and is usually symptomless. Dactylitis is seldom found in the toes. It may exist in both fingers and toes in the same patient. This occurs usually in infants under one year of age.

The typical saber shin is produced by an increase in the cortical bone on the anterior aspect of the tibia. This gives the appearance of bowing and may be differentiated from the bowing in rickets by remembering that in rickets there is bending in the upper two-thirds of the tibia, while in syphilitic osteitis it occurs in the lower two-thirds. A true bowing in this latter region may also

occur, and is considered typical of congenital syphilis.

Pathological fractures may occur, but they are considered most uncommon. Tracy[19] reports one such case, and McLean[10,11] comments upon their rarity.

Syphilitic osteomyelitis is considered by Lovett and Wolbach[9] to be a severe reaction to the infection. This is evidenced by cortical destruction, and with the exception of the coexisting periostitis, may resemble a pyogenic bone infection. These authors also consider that bone proliferation is a response to a mild reaction of the organism to its toxin.

McLean[11] called attention to three types of periostitis: first, existing alone in the absence of coexisting lesions; second, found over an area of rarefaction in the shaft in the presence of an osteochondritis as a healing process, and increasing as the osteochondral lesion recedes; and third, ossifying periostitis producing a thickened, irregular cortex.

Parrot's nodes on the skull are probably due to an old healed periostitis which has become ossified. Hyperostoses of the flat bones are found usually in older individuals, and are considered a manifestation of congenital syphilis. Areas of rarefaction in the inner and outer tables of the skull giving a punched-out appearance are considered diagnostic of lues, and the osteitis, especially on the inner table, is another rather important diagnostic point.

Fig. 1. Osteomyelitis of the congenital syphilitic type. Note the typical periosteal proliferation about the ulna in both upper extremities, plus the typical appearance at the epiphyseal ends of the bones. This periostitis is of the second type in the classification by McLean.

ACQUIRED SYPHILIS

Classification

1. Periostitis
   - A. "Lace-work" type
     - a. Longitudinally arranged
     - b. Transversely arranged
   - B. Ossifying type
2. Osteitis
   - A. Condensing—in trophic joint involvement.
   - B. Hypertrophic—in older cases
   - C. Atrophic—in earlier cases
   - D. Fragmentary—in early trophic involvement
   - E. Nodes on flat bones
3. Arthritis
   - A. Localized
   - B. Trophic
4. Synovitis
5. Osteochondritis
6. Gumma

Many bone and joint conditions in adults which are syphilitic in origin have frequently been mistaken for other diseases. It is comparatively common to find that many adult syphilitics, particularly those who have had the disease for many years, will have a negative blood Wassermann. Roentgenograms of the long bones in many instances will reveal the "lace-work" type proliferation as described by Baetjer and Waters[1] commonly found in this disease. It is interesting to note that in the young adult with acquired syphilis the osseous involvement usually is revealed as atrophic, while in the older individual

who has had the disease for many years the manifestation is always hypertrophic. With the foregoing as a possibility, it may be of academic interest at least to consider that a certain percentage of hypertrophic arthritis with tremendous new bone formation may have an unrecognized syphilitic background. The use of mercury and iodides orally in the form of mixed treatment as a therapeutic test may have an astonishingly beneficial effect where every other form of medication has failed to produce even symptomatic relief.

Fig. 2. Circumscribed rarefying osteitis of the upper ends of both tibiae. The periosteal proliferation is marked together with the characteristic irregular epiphyseal lines and widening of this region.

While it is possible that a history of exposure to syphilis may be denied by the patient and the typical healed scar of the primary lesion may be impossible to find, in the absence of a positive blood Wassermann it may be necessary to make a roentgenogram of the long bones to establish the diagnosis. The typical night pains of the adult syphilitic should be of great assistance diagnostically.

Although the typical roentgenogram of bone syphilis is usually a periostitis, the smaller cancellous bones of the carpus and tarsus may present varying degrees of atrophy or destruction, depending upon the age of the patient and the duration of the disease.

Where excessive destruction takes place without the formation of new bone, in the young adult, the joint is atrophic in appearance. Knaggs[7] shows successive roentgenograms of a shoulder revealing atrophic changes in the first two, and beginning bone deposition in the third. The statement has already been made regarding the manifestation of syphilis in the young adult as being atrophic, particularly in the cancellous bones while in these same bones the manifestation in the older patient is distinctly hypertrophic. This hypertrophic change may be the result of local involvement or may be associated with an involvement of the central nervous system.

The typical Charcot[4,8] joint is not a local syphilitic process but is produced by troph-

ic changes resulting from an involvement of the dorsal columns of the spinal cord. The initial stage of this condition is manifested by hydrops, pain on motion and some tenderness, limitation of motion and occasional involvement of the surrounding structures. The adjacent muscles and the fasciae may be markedly swollen and somewhat tender. Intense, burning night pains in the long bones accompany the early phase. Occasionally in the middle-aged adult the joint is spindle shaped and may give the external appearance of a sarcoma. Contrary to the general belief, the early stage of the Charcot type of joint involvement is painful. This pain is probably due to the remaining uninvolved nerve fibers of the spinal cord.

The impression that is gained on studying a film of a Charcot joint is, first, that there appears to be a condensation of bone in the region of the joint. This density is usually quite marked and the joint outline is distinctly enlarged with a narrowing of the joint space. Second, there is usually more or less hypertrophic lipping on the ends of the bones forming the joint with a distinct evidence of synovitis, and third, the formation of osteochondromatous masses, particularly in the popliteal space. The supra- and infrapatellar regions may be quite distended and hazy. Usually associated with Charcot joints is a distinct periosteal proliferation of the lacy type plus a thickening of the cortical bone.

Occasionally in examining films of knee joints which are somewhat distended with thickened capsules there are found distinct periostitis of the long bones and some hypertrophic changes at the ends of the bones. The joint space may be diminished and hazy with some atrophic areas about the head of the tibia. There may be more or less dense irregular masses on the pos-

Fig. 4. Ossifying periostitis of syphilitic origin. Note the marked irregularity of the cortex of the bones.

Fig. 3. Typical "lace-work" periostitis of acquired syphilis along the shafts of the tibia and fibula. The joint changes are hypertrophic in nature, presenting a hypertrophic arthritis and synovitis together with contracture of the hamstring muscles.

terior aspect of the condyles. In these instances, while it is difficult to indicate the specificity of the arthritis, it is reasonable that syphilis be primarily considered as a potential etiological factor. Many obscure joint pains which have been dismissed as

Fig. 5. Acquired syphilis in young adult. This illustration is typical of the atrophic bone changes in this type of case. There is distinct rarefaction of the anterior aspect of the astragalus and of the scaphoid and the cuneiform bones.

hypertrophic arthritis may, in presenting roentgen evidence such as this, be greatly relieved by the use of antisyphilitic therapy.

Syphilitic osteitis of long bones produces a marked cortical thickening. This thickening is not homogeneous but is a widened, irregular area of alternating condensation and rarefaction so that the roentgenogram has almost the appearance of the osteitis deformans of Paget. However, periosteal proliferation usually is present also. This manifestation may even have the appearance of a pyogenic osteomyelitis.[1] There is, however, a greater amount of bone condensation about the edges of the involved bony area. This same condition may also occur in the flat, cancellous bones of the pelvis, the skull and scapulae.

Gumma of bone has not been a common finding, but it does occur as a manifestation of tertiary syphilis as a localized area of irregular destruction and hyperplasia. Periostitis accompanies this lesion also. Gumma usually appears in the metaphyseal third of long bones but may occur also anywhere within their length, or occasionally may be seen in the flat, cancellous bones of the skeleton.

Syphilitic periostitis is distinctive as a rule, particularly in cases in which the bone involvement has not been too prolonged. Its appearance is that of a fairly delicate, lacy proliferation, and may be very slight or at times exceedingly pronounced. In cases in which bone syphilis has been present for a longer period of time there is a distinct increase in density of this periosteal shadow so that some of the lace-like appearance is lost, but the irregularity in the periosteal proliferation becomes more marked. This may be seen usually about the tibia and fibula, particularly about the junction of the interosseous membrane. This periosteal manifestation is typically luetic and in individuals in whom syphilis may or may not be suspected even in the presence of a negative blood Wassermann reaction, roentgenograms of the long bones may give a suggestion of the specificity of the infection.

While the common conception of syphilitic periostitis concerns its presence parallel to the long axis of the long bones, Pendergrass and Bromer[16] call attention to the fact that this periosteal proliferation may be at right angles to the shaft of the bone and may be seen as a small localized area in roentgenograms in both the anteroposterior and lateral projections. They also note the rarity of periostitis along the posterior aspect of the femur due probably to

arrangements of the muscle attachments in this location.

We realize the discrepancy in the blood Wassermann reactions even in the case of those who give clinical evidence of the primary syphilitic lesion, and a history of inadequate treatment, or in those who have had no treatment at all. It is my belief that in the absence of positive serological findings roentgenograms of the long bones, particularly of the leg, may show the periosteal changes suggestive of syphilis.

Two of the commoner conditions whose manifestations are similar to those of syphilis should be given consideration. These are yaws and syringomyelia. The bone changes in yaws show a great likeness to those in syphilis so that it has been called "frambesial syphilis." Yaws is not regarded as a venereal disease and is considered to be a separate disease entity. From the standpoint of therapy, yaws responds readily to antisyphilitic measures.

In considering the differential diagnosis of trophic joint changes, syringomyelia is frequently confused. This condition is differentiated from syphilis by the distinct loss of the pain and temperature sense, and progressive muscular atrophy accompanied by paralysis. As a general differential point it is better that trophic joint changes existing in the upper extremities be considered of syringomyelitic origin. When the lower extremities are involved, including the lower spine, cerebrospinal syphilis should be considered primarily. However, in the lower extremities when the joint changes are accompanied by muscle wasting and disturbance of the pain and temperature sense this condition is certainly syringomyelia.

Fig. 6. The left ankle reveals marked trophic changes, especially of the anterior tip of the astragalus. The osteochondritis of the astragalus and scaphoid are typical of the moderately advanced trophic changes. In the right astragalus there is a beginning fragmentation of the anterior tip. It is interesting to note that the left astragalus which was roentgenographed in another institution eighteen months ago was reported to be a chip fracture of this region.

TREATMENT

In the treatment of syphilis of the bones and joints as manifested roentgenologically, the physician must realize that in

acquired syphilis the condition, as a rule, is of many years' standing. It is usual that within the so-called period of latency between the second and third stages of the disease, these skeletal changes are noticed. The Charcot joint is distinctly within the tertiary stage, as is gumma. Bone and joint manifestations within this so-called period of latency appear to respond, at least symptomatically, more readily to the time-honored mixed treatment of mercury and iodides orally. Occasionally the symptomatic relief is so marked that patients accuse the physician of giving them narcotics. It is interesting to note that small doses of bichloride of mercury and moderate amounts of the iodide of potassium, when administered over a considerable period of time, will bring about a diminution and occasional disappearance of the periosteal proliferation, together with any osteitis and arthritis of syphilitic origin that may be present. This of course should be reinforced by intravenous and intramuscular injections of the arsenicals and bismuth, so that the patient may have the benefit of complete antiluetic therapy. It has usually been found that this same medication is effective symptomatically in patients with Charcot joints provided the involved joint is properly supported against additional trauma and instability.

In congenital syphilis, however, the use of the arsenicals is definitely indicated and should be continued until the serological reaction is definitely negative. The use of mercury by inunction is distinctly valuable in these instances and should be included in the treatment of such patients. Persistence in treatment, once the diagnosis has been made, is of great importance, as many luetics with bone and joint involvement will receive great relief with these simple measures. No harm can result from the use of mixed treatment as a therapeutic test.

SUMMARY

1. Congenital syphilis is more common than is generally believed.

2. The bone and joint manifestations in congenital syphilis are characteristic.

3. The roentgenological examination of infants and children with obscure bone and joint symptoms may reveal luetic changes which otherwise may be overlooked.

4. Congenital syphilitic bone and joint lesions may be differentiated from those seen in rickets and scurvy.

5. Many adults with acquired syphilis present negative serological findings.

6. The various roentgenological manifestations of acquired bone and joint syphilis are of great value diagnostically.

7. The commonest skeletal lesion in acquired syphilis, particularly in the period of latency, is a typical periosteal proliferation of the more or less lacy type, depending upon the duration of the disease.

8. Syphilitic osteitis in the earlier stage in younger individuals is usually manifested by atrophic changes; in the later stage in older individuals this manifestation is hypertrophic.

9. Syphilitic osteitis and gumma may resemble chronic suppurative infections but the accompanying periostitis of characteristic type is diagnostically important.

10. Trophic joint changes of the Charcot type are manifestations of the tertiary stage of syphilis of the dorsal columns of the spinal cord.

11. The osseous lesions in yaws and the trophic changes in syringomyelia are closely related to those in syphilis, and should be carefully differentiated.

12. Arsenicals for the treatment of congenital syphilis. Mixed treatment orally as a therapeutic test in suspected acquired syphilis may produce gratifying symptomatic relief.

13. Oral medication plus inunctions over long periods of time will produce a disappearance of the osseous and articular luetic lesions.

I wish to acknowledge the kindness extended to me by Dr. S. W. Donaldson, of Ann Arbor, Mich., Dr. Murray E. Goodrich, of Toledo, Ohio, and Dr. John C. Rodick, of New Orleans for the use of their excellent films.

REFERENCES

1. Baetjer, F. H., and Waters, C. A. Injuries and Diseases of Bones and Joints. P. B. Hoeber, New York, 1921, pp. 173–174.
2. Block, I. A System of Syphilis. Edited by D'Arcy Power and J. K. Murphy. Oxford Medical Publications, London, 1908.
3. Butler, C. S., and Biello, J. A. Influence of Ruy Diaz de Isla upon question of American origin of syphilis. *South M. J.*, 1933, *26*, 438–443.
4. Charcot, J. N. *Arch. de physiol.*, 1868, 161.
5. Holt, L. E. Diseases of Infancy and Childhood. D. Appleton & Co., New York, 1911.
6. Jones, J. *Smithsonian Contrib. Knowledge*, 1876, *22*, 45; 61; 65; 85.
7. Knaggs, R. L. Inflammatory and Toxic Diseases of Bone. William Wood & Co., New York, 1926.
8. Lombroso, G. Artropatia tabetica. *Spermentale*, 1885.
9. Lovett, R. W., and Wolbach, S. B. Roentgenographic appearance, diagnosis and pathology of some obscure cases of bone lesions. *Surg., Gynec. & Obst.*, 1920, *31*, 111–131.
10. McLean, S. Roentgenographic and pathologic aspects of congenital osseous syphilis. *Am. J. Dis. Child.*, 1931, *41*, 130–152.
11. McLean, S. Correlation of roentgenographic and pathologic aspects of congenital osseous syphilis, with particular reference to first months of life. *Am. J. Dis. Child.*, 1931, *41*, 363–395.
12. McLean, S. Correlation of roentgenologic picture with gross and microscopic examination of pathologic material in congenital osseous syphilis. *Am. J. Dis. Child.*, 1931, *41*, 607–675.
13. McLean, S. Correlation of clinical picture with osseous lesions of congenital syphilis as shown by x-rays. *Am. J. Dis. Child.*, 1931, *41*, 887; 1128.
14. O'Reilly, A. Syphilis in Bone. Abt's Pediatrics. Vol. V. W. B. Saunders Co., Philadelphia.
15. Parrot, J. Sur une pseudo-paralysie causée par une alteration due systeme osseux, chez les nouveau-resolteints de syphilis héréditaire. *Arch. de physiol. norm. et path.*, 1871–1872. *4*, 314–333.
16. Pendergrass, E. P., and Bromer, R. S. Congenital bone syphilis. Am. J. Roentgenol. & Rad. Therapy, 1929, *22*, 1–21.
17. Shipley, P. G., Pearson, J. W., Weech, A. A., and Greene, C. H. X-ray factors of the bones in diagnosis of syphilis in the fetus and in young infants. *Johns Hopkins Hosp. Bull.*, 1921, *32*, 75.
18. Taylor, R. W. Syphilitic lesions of the osseous system in infants and young children. *Am. J. Obst.*, 1874, *54*, 177–232; *ibid.*, 1875, 559.
19. Tracy, F. E. Advanced osseous syphilis in four-months-old infant. *Radiology*, 1933, *21*, 584–587.
20. Turnbull, H. M. Recognition of congenital syphilitic inflammation of long bones. *Lancet*, 1922, *1*, 1239–1241.
21. Waldeyer, W., and Köbner, H. Beiträge zur Kenntniss der hereditären Knochensyphilis. *Arch. f. path. Anat.*, 1872, *55*, 367–379.
22. Wegner, G. Ueber hereditäre Knochensyphilis bei jungen Kindern. *Virchow's Arch. f. path. Anat.*, 1870, *50*, 305–322.
23. Williams, H. U. Origin and antiquity of syphilis; evidence from diseased bones. *Arch. Path.*, 1932, *13*, 779–814, 931–983.
24. Williams, H. U. Origin of syphilis; evidence from diseased bones. *Arch. Dermat. & Syph.*, 1936, *33*, 783–787.

LUETIC OSTEITIS SIMULATING MALIGNANT DISEASE

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DESCRIPTION of bone pathology resulting from syphilitic infection, as usually given in the literature, is somewhat categorical. As a matter of fact, syphilis affects bone in a greater variety of ways, perhaps, than any other single disease. It causes such tissue to exhibit all but a few of the reactions of which it is capable. Here again syphilis lives up to its reputation as the great imitator.

The three types of luetic bone lesion described in the literature are usually (*a*) congenital, early and late, (*b*) acquired, and (*c*) tertiary. The acquired lesion is manifested in various ways and is classified under four groups, namely (*a*) synovitis, (*b*) arthritis, (*c*) periostitis, and (*d*) osteomyelitis. Late acquired syphilis manifests itself in bone in two general pathological forms, (*a*) chronic inflammatory, (*b*) gummatous.[3] Though it would seem that each of these categories exhibits certain definite pathological, clinical and roentgenographic characteristics, such is not always the case as is well demonstrated in the case here presented.

The pathology of bone syphilis is more or less that of syphilis elsewhere in the body. The spirochetes tend to settle in the vascular tissue of the deeper layers of the periosteum. Perivascular infiltrations occur and a highly cellular granulation tissue is formed around the vessels and in the haversian canals.[4] This gives rise to a thickened, expanding and elevated periosteum which in turn accounts for the early complaint of pain and the early roentgenographic portrayal of hazy thickened periosteum either over a circumscribed area or throughout the shaft.

The granulation tissue erodes the surface of the bone, giving the latter a highly characteristic worm-eaten appearance, best seen in the skull. At the same time the presence of the spirochete tends to stimulate great osteoblastic activity and an abundance of new cells is laid down on the surface and on the walls of the haversian canals, giving rise to extremely dense, heavy bone with irregular enlargement of the shaft.[2] These periosteal thickenings and subperiosteal nodes are recognized in the roentgenogram as localized exostoses. The more severe involvement gives rise to a ripping up of the periosteum with laying down of new cells in laminae either parallel with, or perpendicular to, the axis of the shaft of the bone.[11],[12] This, in the tibia, produces the characteristic saber shin.

The diagnosis of bone syphilis is at times difficult. The evidence is confusing and not always reliable, and, in many instances, the final conclusion must rest on the therapeutic test.

Luetic osteomyelitis may begin as a periostitis and osteitis which has extended to the medulla, with symptoms not unlike those of osteomyelitis of nonspecific origin.[3] The destruction is slow and by direct extension. Pain may or may not be present. There are very slight systemic reactions. Bone changes, as shown by the roentgenogram, are more extensive than one would suspect from the clinical symptoms. Furthermore, it is characteristic that *bone production exceeds bone destruction*.[10]

Roentgenographically, a marked similarity may be present between periosteal syphilis and periosteal sarcoma but the latter shows a rapidly destructive process with little effort at repair. The periosteal proliferation that does occur is in the nature of an irritative reaction with new bone striae perpendicular to the axis of the shaft. In syphilis the new bone formation is more laminated, the laminations lying parallel to the shaft and tending to unite with it at the margins of the process.[10]

In differential diagnosis, Hodgkin's disease may also offer some difficulty. Here

again the lesions, roentgenographically, appear for the most part destructive. There is little or no evidence of bone production. According to Dresser and Spencer[5] Hodgkin's disease involving bone without accompanying involvement of lymph glands, spleen, or liver, has not been observed.

Metastatic malignancy may likewise cause some confusion, but the lesions are almost entirely destructive and involve the medullary cavity early.

In the case here presented certain of the usual clinical, pathological and roentgenographic features were lacking. Moreover, the evidence from these sources created the impression of a malignant destructive process for which a radical therapeutic procedure might have been, but was not, attempted.

REPORT OF CASE

R. W., white male, aged twenty-six, was admitted to St. Elizabeth's Hospital on November 27, 1935. He was confused, unstable, grandiose, showed gross memory defects and expressed ideas of persecution and fear. He exhibited involuntary, fine tremors, hyperactive deep reflexes and Argyll Robertson pupils. An old penile scar was present but the date of this lesion was undetermined. Examination of the blood and spinal fluid gave strongly positive Kolmer and Kahn reactions. The spinal fluid showed 68 cells per cubic millimeter; 45 mg. of protein per 100 cc., and a colloidal gold curve of 5–5–4–3–3–2–1–0–0. He had never received any antiluetic therapy.

Treatment was instituted on January 6, 1936, with a course of 42 centigrams of bismuth subsalicylate. On January 21 he was inoculated with therapeutic quartan malaria and had his first paroxysm of March 1. This was terminated on March 28 after fifteen severe chills. He showed no mental change during this time and suffered no physical ill effect.

On May 2 he developed an acute ulcerative tonsillitis and a multiple furunculosis. Repeated throat cultures revealed the presence of the hemolytic streptococcus over a period of three weeks. Blood culture was negative. Examination of the blood showed normal chemical constituents. The patient was treated symptomatically but did not improve until June 4 when he received a transfusion of 300 cc. of blood. A rapid and complete remission of symptoms followed. During this period no antiluetic treatment was given.

He remained symptom-free until September 28 when he complained of pain over the lower third of the right tibia. This was especially severe at night. On examination the area was found to be swollen, tender and indurated.

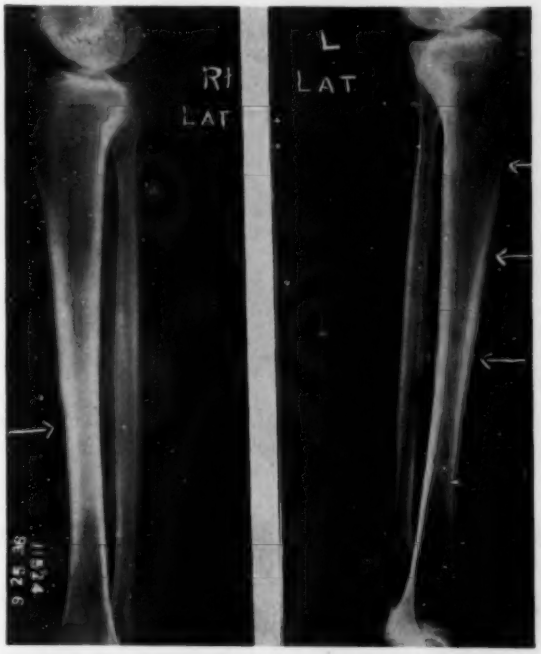

FIG. 1. Arrows indicate some of the lesions discovered at the first examination. Additional areas were shown in posteroanterior views in this and in subsequent examinations.

Roentgenographic examination showed multiple destructive areas, varying in size and shape, in both tibiae and fibulae (Fig. 1). The margins of these lesions were not marked by any increase in density and they were limited entirely to the cortex with no evidence of periosteal or medullary involvement and without any roentgenographic evidence of bone production.

The lesions were more numerous and more extensive in the left tibia in spite of the fact that the complaint and clinical findings centered about the right tibia. The lesions were more extensive than the symptomatology indicated. The roentgenologist was of the opinion that the pathology represented was a malignant one and, in spite of the fact that it was bilateral, probably a multiple primary sarcoma. Roent-

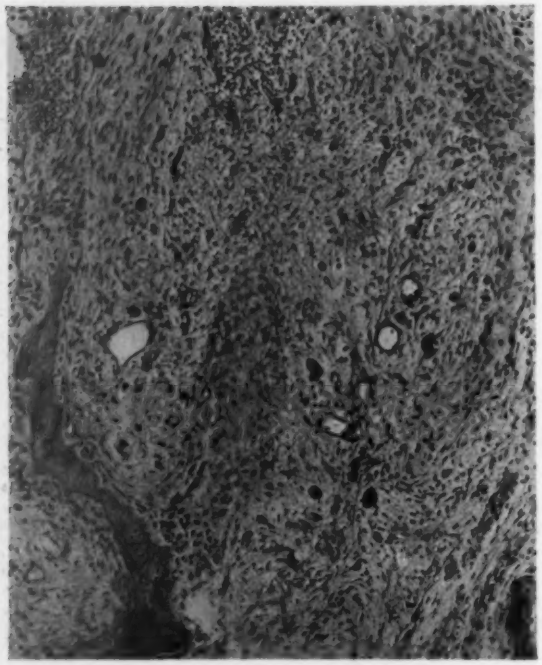

FIG. 2. Low power magnification showing giant cells, foci of lymphocytes, thin-walled blood vessels, and area of hemorrhage.

genographic studies of the remainder of the skeleton were entirely negative.

On the basis of this report and the clinical picture a biopsy was done on October 7. The specimen was taken from the anterior surface of the right tibia, the site of the initial swelling. The pathologist reported that the tissue consisted of a number of bone lamellae, some of which were outlined by osteoblasts. The space between the lamellae was occupied by loose connective tissue containing many thin-walled blood vessels. A few small hemorrhages were evident. Lying in this granulation tissue were several small foci of lymphocytes. A number of giant cells were scattered throughout the fibrous stroma, one having as many as eight nuclei (Fig. 2). Usually the number of nuclei was three or four. The giant cells were of the foreign body type. Mallory's connective tissue stain showed that in some places the fibrils were gathered into closely interlacing strands surrounding large vesicular nuclei with prominent nucleoli. Another area presented an altogether different appearance (Fig. 3). The loose reticulum was markedly infiltrated with plasma cells, polymorphonuclear leukocytes and large mononuclear cells with round or oval vesicular nuclei resembling those of the reticulo-endothelial system. Carcinomatous metastasis was definitely ruled out. Although the process was one of inflammation involving periosteum in the form of fibrosis and osteophyte formation, the compact bone was destroyed and replaced by granulation tissue. The cellular polymorphism, the intense reticulo-endothelial hyperplasia and the presence of giant cells seemed to constitute together a fairly characteristic syndrome of Hodgkin's disease. Stains for spirochetes were negative.

During the first week in October the patient suffered a recurrent ulcerative tonsillitis, showed gradual loss of appetite and weight and developed progressive weakness. This time he failed to respond to a blood transfusion. Repeated blood studies revealed a hemoglobin varying from 60 to 64 per cent; an erythrocyte count between 3 and 4 million; a white cell count from 10,000 to 14,000 with 22 per cent lymphocytes, 62 per cent polymorphonuclear cells and an occasional myelocyte. Sedimentation tests varied between the diagonal and

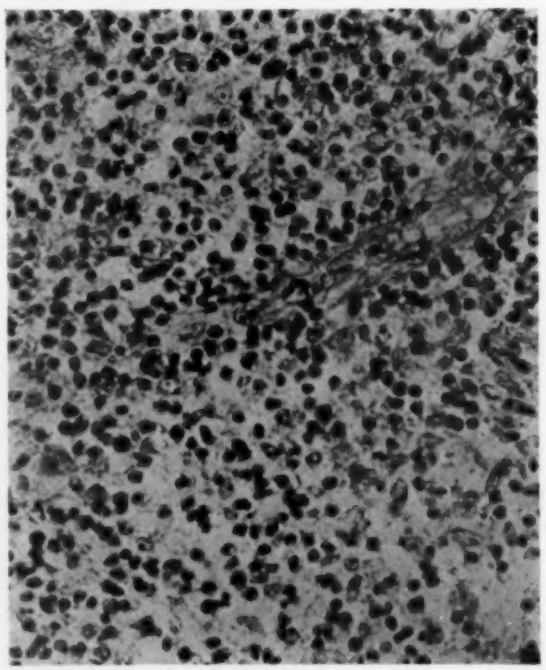

FIG. 3. High power magnification of another field showing loose reticulum infiltrated with plasma cells, polymorphonuclear leucocytes and large mononuclear cells.

vertical curves. The urine reports were always within normal limits except that on one occasion a trace of Bence-Jones protein was reported. A roentgenogram on November 7 showed further destruction in the lesions of the tibiae and fibulae, their continued limitation to the cortex and the absence of any evidence of bone production (Fig. 4).

Up to this point, roentgenographically and pathologically, the picture was one of malignant involvement of bone. However, the clini-

Fig. 4. Showing progressive destruction taking place six weeks after initial examination.

cal picture and the history of lues warranted an antiluetic therapeutic test. On November 9 active antiluetic therapy was instituted and 1.3 grams of bismuth subsalicylate and 4.65 grams of neoarsphenamine were given during the subsequent eight weeks.

Clinical improvement was dramatic during the first two weeks and by December 30 the patient was entirely free of all clinical symptoms. As can be seen in Figure 5, there was no evidence of improvement roentgenographically. However, as seen in Figure 6, made on January 21, the lesions showed definite improvement and, as shown in Figure 7, made on March 3, healing was almost complete, with the first real

Fig. 5. No improvement in roentgenographic appearance seven weeks after beginning of antiluetic therapy, although all clinical symptoms had disappeared.

Fig. 6. Showing first sign of improvement, roentgenographically, ten weeks after beginning of treatment.

evidence of bone production, in the form of periosteal thickening in the fibulae.

From February 15 to March 29 the patient received 10 grams of tryparsamide and 65 centigrams of bismuth subsalicylate. On March 23 the Kolmer reaction of the blood and spinal fluid was weakly positive, the latter showing also a protein content of 20 mg. per 100 cc., no

FIG. 7. Showing healing almost complete and first signs of bone production, indicated by periosteal thickening of the fibulae, approximately seventeen weeks after treatment was instituted.

cells and a colloidal gold curve of 2–2–2–3–2–1–0–0–0. He has gained 10 pounds in weight and is entirely symptom-free. Antiluetic therapy is being continued.

As was demonstrated, the final diagnosis in this case rested on the therapeutic test. The microscopic picture was certainly atypical and the roentgenographic evidence was not only atypical but contrary to the usual appearance of luetic osteitis, mainly because of the purely destructive character of the lesions without any evidence of bone production, and also their limitation entirely to the cortex of the bone. A portion of the microscopic picture suggested the possibility of Hodgkin's disease involving bone and the roentgenographic appearance seemingly could be better explained on this basis; but there was complete absence of involvement of the liver, spleen and lymph glands.

The effect of arsenic on Hodgkin's disease is recognized but in view of the above we feel that the case was solely one of luetic osteitis which responded dramatically to antiluetic treatment, both clinically and roentgenographically.

It is of interest to note that the bone lesions in this case were associated with general paresis, an occurrence which is rare according to Wasserman and Goodman[14] who found that of 250 cases of late mucocutaneous and osseous (benign late) syphilis, only 3, or 1.2 per cent, had general paresis.

SUMMARY

1. Lesions of bone syphilis may occur in which the roentgenographic evidence is solely of bone destruction with no suggestion of the characteristic bone production.

2. Roentgenographically, luetic bone lesions may show involvement of the cortex without demonstrable periosteal or medullary changes.

3. Cases of doubtful bone pathology should be given the benefit of therapeutic antiluetic test before more radical procedure is adopted.

REFERENCES

1. BARTHÉLEMY, R. Traumatic syphilis of bones and joints. *Urol. & Cutan. Rev.*, 1930, *34*, 14–18.
2. BOYD, W. Surgical Pathology. Second edition. W. B. Saunders Co., Philadelphia, 1930.
3. CLARK, J. J. Diagnosis of syphilitic bone lesions. *J. Med. Ass. Georgia*, 1927, *16*, 369–373.
4. DELAFIELD, F., and PRUDDEN, T. M. A Text-Book of Pathology. Fifteenth edition. William Wood & Co., 1931, p. 1087.
5. DRESSER, R., and SPENCER, J. Hodgkin's disease and allied conditions of bone. AM. J. ROENTGENOL. & RAD. THERAPY, 1936, *36*, 809–815.

6. Duggan, L. B., and Bromberg, L. Syphilis of bone resembling osteitis deformans. *Am. J. Syph.*, 1931, *15*, 538–543.
7. Galvin, A. H. Syphilis of osseous system. *Am. J. Syph.*, 1928, *12*, 187–193.
8. Lamb, H. W. Bone and joint syphilis. *J. Maine M. Ass.*, 1929, *20*, 47–52.
9. Moore, J. E. Modern Treatment of Syphilis. C. C. Thomas, Springfield, Ill., 1933, p. 269.
10. O'Reilly, A. Syphilis of osseous system. *J. Missouri M. Ass.*, 1932, *29*, 577–580.
11. Peters, J. J. Syphilis of bones. *J. Nat. M. Ass.* 1929, *21*, 48–50.
12. Sante, L. R. Radiographic manifestations of syphilitic disease of bone. *Am. J. Syph.*, 1928, *12*, 510–516.
13. Speed, J. S., and Boyd, H. B. Bone syphilis. *South. M. J.*, 1936, *29*, 371–377.
14. Wasserman, H., and Goodman, M. J. Results of treatment in late mucocutaneous and osseous (benign late) syphilis. *Am. J. Syph. & Neurol.*, 1934, *18*, 458–467.

ROENTGEN FINDINGS IN MORVAN'S TYPE OF SYRINGOMYELIA

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IN 1837, the name of syringomyelia was first used by Ollivier[3] to designate any canal or cavity in the spinal cord. A Breton physician, named Morvan,[17] in 1883 described a chronic affection characterized clinically by neuralgic pains, tactile and thermal anesthesia, analgesia and destructive felons. Morvan's disease, as this affection was called, is no longer considered a separate condition, but merely a type of syringomyelia, which may be defined pathologically as a disease of the spinal cord characterized by the development of gliomatous tissue in the central parts with the formation of cavities. Necropsies show, however, that cavities are not always formed, and then some authors prefer to use the term spinal gliosis. Clinically, all classes of syringomyelia including the amyotrophic, spastic, Morvan's and spinal tumor types, are manifested by the slow progressive development of muscular atrophy with peculiar disturbances of sensation and of vasomotor function.

Usually, in syringomyelia, there is a characteristic loss of temperature and pain sense, while tactile sensation is retained. In Morvan's type, tactile anesthesia is added. The involvement of the crossed pain and temperature fibers in the anterior commissure, and sparing of the tactile fibers in the posterior columns, except when involved in Morvan's type, constitutes the physiological explanation. Involvement of the motor cells of the anterior horns may produce marked atrophies, as of the small hand muscles. Involvement of the pyramidal tracts may produce a definite spastic paraplegia. Morvan's type is a cervical form with trophic changes of the bones of the fingers and hands—a sclerodactylia. The various joints of the upper extremities, the spine and thorax may also be involved in these trophic changes. Felons, mutilation of the fingers, soft tissue swelling, dryness of the skin, loss of nails, atrophy of the phalanges, ulcers devoid of the slightest pain, are typical of this form. Boils, abscesses and other infections which heal poorly, producing extensive scars and deformities occur. Variations of Raynaud's syndrome, minus pain, are common.

It seems probable that all of the bone changes which occur in syringomyelia are more common than are generally recognized. Standard roentgenologic textbooks casually mention the subject in their paragraphs on Charcot's joints. Strangely enough, texts professing to cover bones and joints, in many instances omit the subject entirely. Baetjer and Waters,[2] in their excellent work, offer a notable exception to this rule. Neustaedter,[13] Ashhurst,[1] and Buzzard[3] are also exceptions to the majority, who omit the roentgen aspects entirely in dealing with this subject.

Since 1868, tabetic arthropathies have commonly been called after Charcot,[4] but as he, at that time, described the changes as occurring in syringomyelia as well, properly speaking "Charcot's joints" should include both forms. Numerous authors hasten to point out that the tabetic form occurs more often in the lower weight-bearing joints, while the joints of syringomyelia are more common in the upper extremities. In spite of these repeated observations, which appear so monotonously throughout the literature, a terse diagnosis of "Charcot's joint" rendered by a roentgenologist is often sufficient to condemn a patient to a syphilis clinic with its almost endless intravenous and intramuscular ministrations. An examination of the other bones of the body and information concerning the history and clinical findings, if possible, may often help the roentgenologist to be more specific in his diagnosis, as

tabes and syringomyelia are not the only conditions responsible for the arthropathy described by Charcot. The busy clinician, however, recognizing the superiority of roentgenograms of the long bones to the blood Wassermann reaction in the diagnosis of congenital syphilis in a baby, applies the same reasoning in a common tabetic conception of Charcot's joint. He is apt to accept the roentgen diagnosis and neglect further serologic and neurologic data. The problem is not nearly so simple. Theories and speculation as to the etiology of nervous arthropathies began directly with Charcot's description and are still very much in process, and certainly are not at all complete.

Charcot believed that the joint changes resulted from a destruction of trophic fibers in the cord produced by the tabes or syringomyelia. Increased trauma in the ataxic individual, other cord lesions as spina bifida, myelitis, postencephalitic changes, direct injury to the cord as in stab wounds, and peripheral nerve injuries have been described as causes of the same type of arthropathy by Duncan,[6] Eloesser,[8] Philips and Rosenheck,[15] Wile and Butler,[19] Ashhurst,[1] and others.

Eloesser's[8] experiments, in which, by cutting the posterior nerve roots (the sensory fibers) in cats, he produced bone and joint lesions corresponding to those found in tabes dorsalis, are outstanding. Sixty-nine cats were used, 42 were observed for periods ranging from three months to over three years, and they were all healthy animals so that the tabetic fractures and arthropathies produced by severing the posterior nerve roots from the limb to be involved, could not be ascribed to syphilis or other infectious causes. Severing the posterior roots caused no atrophy of bone, and nothing in the experiments gave proof of the existence of trophic nerves. The studies, which were complete in surgical, chemical, pathologic and roentgenologic detail, end with the observation that "trauma in a limb rendered anaesthetic and analgesic experimentally leads to grotesque lesions of the bones and joints, which are in every way the counterparts of tabic fractures and arthropathies; trauma and lack of the warning sense of pain are the cause of most tabic bone and joint lesions." Some of the cases showed trophic ulcers and soft tissue changes associated with the arthropathies similar to those seen in Morvan's type of syringomyelia.

Cotton,[5] to quote an opposite, and what appears to be a small minority opinion, states that Charcot's joints should be considered as syphilitic arthritides.

Recently case reports of bone and joint findings in syringomyelia have appeared. Pokorny,[16] in 1932, detailed a case with bone changes in the shoulder and carpals. Fried,[10] in dealing with a case, quotes Baelestra's work on bone and joint findings done in 1931. Zimmer,[20] in 1935, reported 3 separate cases, one with shoulder arthropathy, a second with vertebral scoliosis, and a third showing atrophy of the terminal phalanges of the fingers. This latter paper treated the therapeutic aspects of the subject at length and in this respect is typical of most of the extensive foreign literature on syringomyelia. O'Brien[14] recently published a report of 30 cases of his own and a bibliography of sixty papers mostly foreign, tending to demonstrate the value of roentgen therapy in alleviating the bizarre symptoms and controlling or arresting the condition. This therapeutic phase of the subject is not the concern of this paper. Warfield[18] described a case showing an arthropathy of an elbow and his paper, entitled "Charcot's disease of the elbow joint," illustrates the difficulty in tracing down the diagnostic roentgen literature of syringomyelia, which is hopelessly bound up in hundreds of articles on Charcot's joints.

In consideration of the confusion still apparently prevalent, it seemed worth while to present a case of Morvan's type of syringomyelia, diagnosed by roentgen methods alone, undiagnosed clinically for more than ten years. In fact the first diagnosis attached to the patient's chart on the outpatient side, namely a "Charcot's

joint" did not materially help the patient, as it might have, if an attempt had been made to further qualify the type of arthropathy by roentgen examination of the other bones of the body. The record is admittedly incomplete, partly due to the patient's low mental status, and partly to the system which permits brief observations, at irregular intervals by numerous students and visiting physicians. The omission of roentgen therapy and spinal fluid examinations were due to circumstances quite beyond the radiologist's control.

CASE REPORT

L. L. B., female, colored, single, aged forty-five, a maid by occupation, was admitted to the Outpatient Medical Clinic on January 19, 1925. Her chief complaint was pain in the left knee of four days' duration. This was continuous and throbbing in character. It began at night and had increased in severity. The family history disclosed her father and mother unknown to her, while two sisters were living and well. The past medical history showed several diseases of childhood. She denied venereal infection. Occasional sore throat, ear and headaches had been noted. The menses were regular.

Physical examination showed a mentally subnormal middle aged negro woman, well developed and fairly well nourished. The pupils reacted to light and accommodation. The only positive findings were an inflamed right tonsil, pyorrhea, carious and several missing teeth. The left knee joint was rigid, painful on external palpation, especially on the outer aspect. It was impossible to flex the knee without acute pain. The blood Wassermann reaction and urinalysis were negative. Salicylates were prescribed. No diagnosis was recorded.

April 14, 1931. Patient returned to the Outpatient Surgical Clinic. Osteomyelitis was suspected at the end of the left ring finger. The finger was dressed and patient referred to the hospital.

April 16, 1931. On the Surgical Service of the hospital the history shows that the left ring finger started to swell on April 10, 1931, that it became painful and a sloughing lesion which drained pus was noted.

Roentgen examination of the left hand on April 17, 1931, showed: "There is evidence of a small amount of bone destruction at the tip of the distal phalanx of the fourth or ring finger. Some soft tissue swelling is seen in this region. There has been some bone destruction of the distal half of the distal phalanx of the thumb. A small detached piece of this phalanx is free in the soft tissue under the nail. Nothing else of clinical significance is noted and no atheroma of the blood vessels is seen."

On April 20, 1931, the blood Wassermann was negative. The hemoglobin was 80 per cent (Sahli), red blood cells 3,850,000, white blood cells 10,800, polymorphonuclears 77 per cent, lymphocytes 22 per cent, and monocytes 1 per cent. The urine was negative.

The following excerpts from the hospital record, at that time, are significant in the light of the following events. "The left ring finger is sloughing and the entire left hand appears slightly swollen and feels numb." "Similar affection of the thumb years ago." "The reflexes were normal."

The temperature, pulse and respiration were normal. Warm magnesium sulphate wet dressings were used and the patient was discharged May 20, 1931, as improved with a final diagnosis of "osteomyelitis of the left ring finger."

September 14, 1933. The blood Wassermann was negative.

September 23, 1933. The patient was seen in the outpatient surgical clinic complaining that she was losing the nails of her fingers and that she first noticed this tendency about three years before. She complained of pain on the ventral surfaces of the fingers of about four years' duration. One week previous to her visit, the skin on the finger cracked and tissues parted leaving ulcerated areas all the way down to the muscles. There was no drainage and no tendency to heal. Examination showed blunted fingers, no nails and deep furrows and scratches on the fingers of both hands. The urine was negative. No diagnosis was recorded.

October 7, 1933. At the outpatient medical clinic, the senses of pain, temperature and touch were present in the fingers. The patient could not differentiate between a sharp and a dull point in the fingers. The reflexes in the extremities were exaggerated. No diagnosis was recorded but the patient was referred to the neurologic service.

October 19, 1933. The outpatient neurologic clinic apparently considered the possibility of syringomyelia and referred the patient to the hospital for spinal fluid examination. Pain and

temperature sensation in the hands was present. There were no abnormal reflexes. The nails and skin of both hands were atrophying. There was an ulcer between the left thumb and index finger. No diagnosis was recorded.

June 13, 1934. At the surgical dispensary a chronic discharging ulcer at the base of the left thumb was dressed.

April 4, 1935. Outpatient medical clinic. There was a generalized swelling of the left arm which was brawny on palpation. There was no pitting. Just below and outside the left shoulder, there was a rounded swelling which had a cystic appearance. Patient was unable to use her left arm. There was no pain or soreness. She was referred to the orthopedic clinic but apparently never arrived there.

September 9, 1935. Outpatient medical clinic. There was remarkable brawny swelling of the tissues around the left shoulder girdle which seemed larger than before. Roentgen examination of this shoulder was requested and potassium iodide prescribed.

Roentgen examination on September 9, 1935 (Fig. 1): "There was complete disorganization of the left shoulder joint, pathologic fracture of the head and neck of the humerus, numerous large irregular masses of bone and calcified material scattered throughout and tremendous soft tissue swelling typical of a Charcot's joint."

September 14, 1935. The roentgen diagnosis of Charcot's joint was not accepted by the medical service without protest as they admitted quite frankly that they had expected an "osteogenic sarcoma" and they took the films to two well-known roentgenologists who offered no other diagnosis than "Charcot's joint." The medical service having checked the roentgen diagnosis to their own satisfaction hastened to recommend the patient to the syphilis clinic for treatment. This procedure was apparently based on the common tabetic conception of a "Charcot's joint." This was done in spite of five negative blood Wassermann reactions, no positive Wassermann reactions, and lack of positive clinical or neurologic signs or symptoms of syphilis or tabes, covering a ten-year period. The patient received several of the routine intravenous arsphenamine and intramuscular bismuth salicylate injections with no note of any apparent change in the condition of the patient.

Fig. 1. Charcot's joint of the left shoulder showing widespread disorganization, fracture, bony and calcific fragments.

October 8, 1935. The patient was found lying on the street in a semi-stuporous condition, was picked up by the city ambulance and admitted

Fig. 2. Left hand showing atrophy of most of terminal phalanges with flexion of distal interphalangeal joints giving the typical "claw hand" of Morvan's type of syringomyelia.

to the surgical service of the hospital. The upper left extremity was swollen from the shoulder to the fingers but was not especially tender on palpation. There was a felon of the left thumb which drained pus freely. The patient had lost the tips of the left thumb, index, middle and ring fingers and the right thumb. The temperature was 102.4°F., hemoglobin 78 per cent (Sahli), red blood cells 4,370,000, white blood cells 21,800, polymorphonuclears 85 per cent, lymphocytes 15 per cent. The urine was negative. The blood Wassermann reaction was negative (this latter following the intensive antiluetic therapy above mentioned).

On October 10, 1935, the patient was referred to the roentgen department for examination of the left thumb (Figs. 2, 3 and 4). The report follows: "The left thumb showed the distal half of its terminal phalanx to be missing, apparently due to atrophy. The distal end of the remaining bone was irregular with a tiny oval area of rarefaction but no sequestrum. The periosteum was not elevated. There was no osteomyelitis in the other remaining phalanges. Incidentally, the fingers of both hands were examined for comparison and there was atrophy of the terminal ends of the left index, middle and ring fingers and right thumb, all in their distal phalanges with soft tissue swelling of the left hand generally."

"These findings in conjunction with the widespread destruction and disorganization previously described in the left shoulder as a Charcot's joint force us to further qualify our diagnosis."

"Conclusion: Syringomyelia (Morvan's type)."

Additional roentgen examinations of all the

Fig. 3. Additional projection of left thumb showing atrophy of distal half of distal phalanx with irregularity of distal portion of remaining bone.

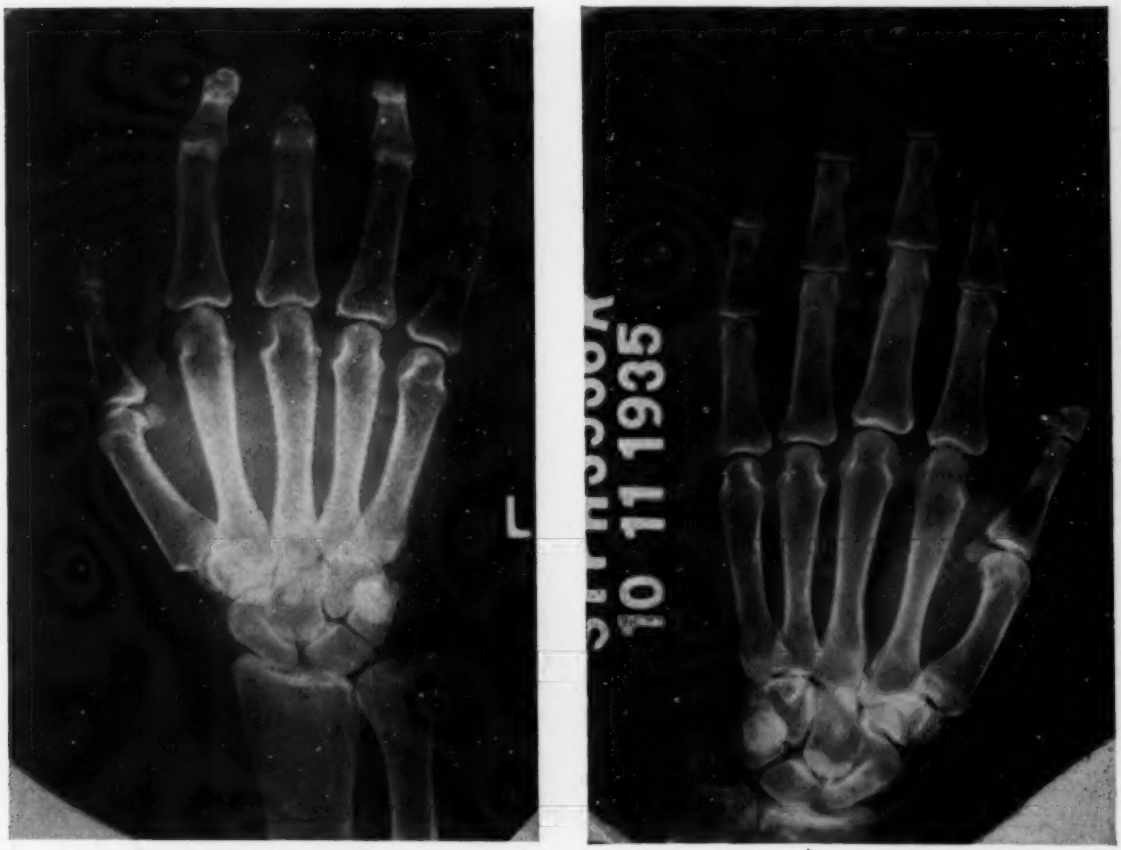

Fig. 4. Roentgenograms of both hands showing atrophy of the distal half of right thumb in addition to the "claw hand" on left.

bones of the body from the skull to the feet were completed during October by the roentgen department, for their own interest only.

The positive findings consisted of a very slight dorsolumbar scoliosis with the convexity to the right. There was rather marked lipping of the lower lumbar vertebral bodies (Fig. 6).

The thorax showed old fractures of the posterior left sixth, seventh, eighth, ninth and tenth, and the right eleventh ribs, with approximate alignment of the fragments, abundant callus and firm bony union. The left sixth, seventh and eighth ribs show best on the print (Fig. 5).

The left wrist, carpal and carpometacarpal joint spaces were narrowed, suggesting cartilaginous destruction. There was extensive irregular adjacent soft tissue density and swelling (Fig. 8).

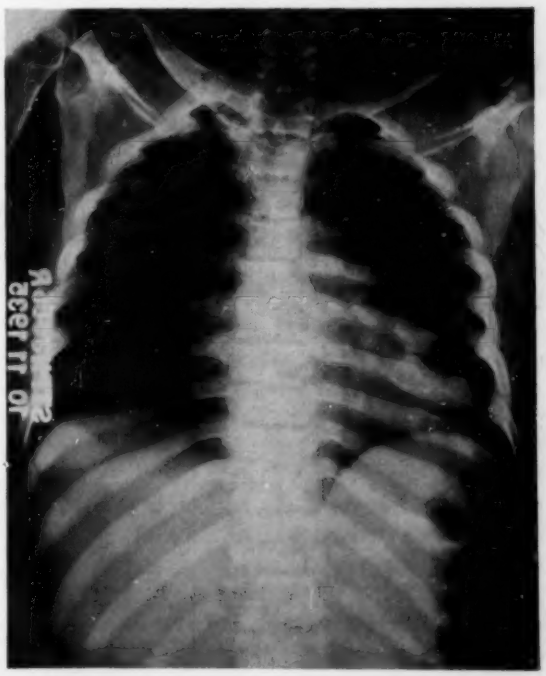

Fig. 5. Thorax showing old fractures of the left sixth, seventh and eighth ribs posteriorly with abundant bony union.

Fig. 6. Dorsolumbar scoliosis with marked lipping of the vertebral bodies, quite usual in syringomyelia of Morvan's type.

The left elbow joint space was preserved but there was a large irregular bony spur at the coronoid process of the ulna (Fig. 7).

The above plus the marked arthropathy of the left shoulder (Fig. 1) and atrophy of the terminal phalanges of the fingers with the somewhat typical "claw" position of the phalanges completed the positive findings in the case.

Deep roentgen therapy of the spinal cord was cautiously suggested as a possible means of controlling or at least retarding the process of the disease.

Following the roentgen diagnosis, a member of the surgical service was kind enough to write upon the patient's chart the following: "Roentgenograms of hands show bone destruction of terminal phalanges (trophic change). Patient has loss of temperature sense in hands to gross test. These findings with scoliosis, arthropathy of left shoulder and additional findings of several old fractured ribs fit in with a revised diagnosis of Morvan's type of syringomyelia, which was first suggested by the roentgen department."

October 17, 1935. The infection of the left hand became worse and extended up the left forearm. Conservative surgical measures with elevation of the part and wet dressings were of no avail. Multiple incisions were made over the left hand and lower forearm and numerous pockets of pus were found. Five drains were inserted.

November 8, 1935. There was no improvement in the infection. A guillotine amputation of the lower seven-eighths of the left forearm was performed. The stump was not closed and and flaps were everted. The pathologic report by Dr. A. C. Broders follows: "The hand is swollen and the skin is partially desquamated. There is a sloughed area on the medial side of the index finger exposing the bone. The skin is thickened and there is necrosis of the subcutaneous tissue. The dorsal palmar space is filled with an inspissated pus and there are similar collections in other places. The wrist joint shows a loss of cartilage and easy disarticulation of the bones is noted. The thumb tip shows an irregular loss of bone with only soft

Fig. 7. Narrowing of the joint spaces at the wrist, and spurring of the coronoid of the ulna, on the left.

Fig. 8. The claw hand, with atrophy of terminal phalanges, destruction of wrist joint cartilage, probably due to infection superimposed on trophic changes, with irregular soft tissue densities of marked infection.

yellowish material remaining.

"Purulent osteomyelitis and cellulitis with a fairly marked necrosis of bone and soft tissues. There are areas of bone in which osteoblasts are active and other areas in which they are not."

December 1, 1935. There was granulation tissue over the stump and the flaps were covered with healthy granulation tissue.

December 28, 1935. The patient was discharged. There was no drainage from the stump.

COMMENT

Clinically, trophic changes are observed with equal incidence in peripheral and central diseases of the nervous system. According to Duncan,[6] there is no unequivocal proof for the presence of specific trophic nerves, but all efferent nerves exert an influence on the nutrition of the tissues supplied to them. He further states that the influence exerted by the afferent nerves is not less important. Most lesions of the afferent nerves are related to disturbances of so-called trophic function. His conception is that when the efferent nerves are destroyed, the characteristic result is atrophy; but when the afferent nerves are destroyed the result may be either atrophy or hypertrophy. He presents a case of a lumberman, aged forty-two, on whom a log fell, injuring the posterior roots of the lower cervical and upper thoracic segments. The anterior motor roots were unaffected and the patient was able to use his arm. His right upper extremity was swollen from shoulder to wrist. The shoulder joint space showed tremendous widening. The carpals were disorganized, the radius and ulna hypertrophied. There was no pain and definite anesthesia. From a

purely roentgen diagnostic standpoint, the findings were very similar to the case of syringomyelia we have just reported. Eloesser's experiments, which have been mentioned, also serve as a warning at any attempt to find anything pathognomonic in our combination of findings.

In 1925, our patient had originally come under observation for an acute condition of the left knee. Roentgen examination in 1935 showed no evidence of recent or old pathologic change in the knees or any bones of the lower extremities.

In 1931, the report of the roentgen examination of the left hand reflects the influence of the roentgen literature then current on the roentgenologist's processes of thought. Not long before, Kornblum[12] had described bone changes in Raynaud's disease and Edeiken[7] had published his signs thought to be pathognomonic of scleroderma. Various pathologic entities which produce changes in the terminal phalanges were considered by these authors. In this case, the examiner then on duty closed his report with the statement that "nothing else of clinical significance is noted and no atheroma of the blood vessels is seen." Any large roentgenologic service yields occasionally cases showing atrophy of the terminal phalanges of the fingers in osteomyelitis, diabetic gangrene, gout, Buerger's and Raynaud's disease and the literature now and then reports the same findings in leprosy, syringomyelia, psoriasis, ainhum (in the toes) and other rare conditions.

In 1935, the roentgenologist rendered a diagnosis of "Charcot's joint" of the shoulder in a patient clinically thought to have an osteogenic sarcoma. The matter would have been forgotten in the maze of routine, if the roentgen diagnosis had not been so frankly questioned by the medical service. This led to some reference work to confirm our already expressed opinion. The re-examination of the patient for an entirely different complaint namely "osteomyelitis of the thumb" would spur the most lethargic roentgenologist to make an even more positive diagnosis, especially when the clinicians had displayed their sole tabetic conception of Charcot's arthropathy.

SUMMARY

1. A case of Morvan's type of syringomyelia, presenting the classical symptoms and findings, is reported.

2. The roentgen findings include atrophy of the terminal phalanges of the fingers, trophic changes in the joints of the upper extremities, scoliosis and thoracic deformities in the nature of pathologic fractures.

3. The roentgen examination of all the bones of the body is often invaluable, when the examination of a single part appears at all inconclusive.

4. A so-called "Charcot's joint" merits clinical and laboratory examinations for diseases and injuries of the peripheral as well as the central nervous system.

5. Atrophy of the terminal phalanges of the fingers occurs in, and is obviously not pathognomonic of, syringomyelia, leprosy, gout, psoriasis, osteomyelitis, diabetic gangrene, frostbite, scleroderma, Buerger's and Raynaud's disease.

REFERENCES

1. Ashhurst, A. P. C. Surgery: Its Principles and Practice. Fourth edition. Lea and Febiger, Philadelphia, 1931, p. 493.
2. Baetjer, F. H., and Waters, C. A. Injuries and Diseases of the Bones and Joints. Paul B. Hoeber, Inc., New York, 1921, pp. 325–233; 302; 304–305.
3. Buzzard, E. F. Syringomyelia. In: Osler, William. Modern Medicine. Lea and Febiger, Philadelphia, 1910, Vol. VII, pp. 214–230.
4. Charcot's disease of the knee. *Ann. Surg.*, 1897, *25*, 489–490.
5. Cotton, F. J. Surgical aspects of Charcot joint and other syphilitic bone and joint lesions. *Ann. Surg.*, 1920, *72*, 488–493.
6. Duncan, J. H. Neuropathic arthritis. *J. Am. M. Ass.*, 1922, *79*, 1987–1989.
7. Edeiken, L. Scleroderma with sclerodactylia. Am. J. Roentgenol. & Rad. Therapy, 1929, *22*, 42–44.
8. Eloesser, L. On the nature of neuropathic affections of the joints. *Ann. Surg.*, 1917, *66*, 201–207.
9. Eloesser, L. A sign occurring in cases of tabes

complicated by Charcot joints. *J. Am. M. Ass.*, 1921, *77*, 604.

10. FRIED, H. Illustrative case of syringomyelia treated with roentgen rays. *Radiology*, 1934, *23*, 705–709.
11. HOLMES, G. W., and RUGGLES, H. E. Roentgen Interpretation. Fourth edition. Lea and Febiger, Philadelphia, 1931, p. 165.
12. KORNBLUM, K. Bone changes in Raynaud's disease as revealed by the roentgen ray. AM. J. ROENTGENOL. & RAD. THERAPY, 1929, *21*, 448–452.
13. NEUSTAEDTER, M. Textbook of Clinical Neurology for Students and Practitioners. F. A. Davis & Co., Philadelphia, 1929, pp. 263–274.
14. O'BRIEN, F. W. Roentgen therapy in syringomyelia. *Radiology*, 1935, *24*, 16–21.
15. PHILIPS, H. B., and ROSENHECK, C. Neuroarthropathies; consideration of etiology and general characteristics. *J. Am. M. Ass.*, 1924, *82*, 27–29.
16. POKORNY, L. Osteo-arthritische Form der Syringomyelie. *Röntgenpraxis*, 1932, *4*, 349–352.
17. TYSON, J. Practice of Medicine. P. Blakiston's Son & Co., Philadelphia, 1896, p. 883.
18. WARFIELD, C. H. Charcot's disease of the elbow joint. AM. J. ROENTGENOL. & RAD. THERAPY, 1933, *29*, 530–532.
19. WILE, U. J., and BUTLER, M. G. Critical survey of Charcot's arthropathy. *J. Am. M. Ass.*, 1930, *94*, 1053–1055.
20. ZIMMER, E. A. Die Syringomyelie unter Berücksichtigung ihrer röntgenologischen Diagnostik und therapeutischen Beeinflussung. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1935, *51*, 247–260.

CONGENITAL ABSENCE OF THE MIDDLE OF THE ESOPHAGUS*

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CONGENITAL malformations of the esophagus are by no means extremely uncommon. They usually manifest their presence within a few days of birth, almost all proving rapidly fatal.

According to Thomas, the first case was observed by Thomas Gibson in 1696. A case was reported by Martin in 1825. McKenzie found 63 instances up to 1884. Plass found 204 cases up to 1919, but Hirsch could only verify 146 cases up to 1921. Reynolds and Morrison state that only one case appeared in 16 years at Bellevue Hospital.

CLASSIFICATION

Though the types may vary in minor details, the picture is generally uniform as far as the esophagus is concerned. The upper portion ends in a blind cul-de-sac at varying levels near the tracheal bifurcation, while the lower portion begins in a blind pocket just proximal to the cardiac sphincter of the stomach. The blind ends may or may not be connected by a fibrous or muscular cord or band, and there may be an associated tracheal fistula. There may be other anomalous findings in the gastrointestinal tract. The upper blind end generally shows some dilatation.

Congenital absence of the middle of the esophagus occurs: (1) With esophagotracheal fistula. Hirsch found a fistula present in 96 out of 136 verified cases. This is recognized as the most common form. (2) With esophagotracheal fistula and with some fibrous or muscular connection between the two blind pockets. Hirsch found this present in 51 cases out of 136. There was no lumen in the connecting tissue. (3) Without a fistula and without any connecting tissue between the blind ends. No figures are available but it is the rarest of the forms. (4) With associated anomalies and with or without fistula. This was found in 59 out of 94 cases. The most common association was atresia ani, being noted in 24 instances. Meckel's diverticulum and atresia of the duodenum also occur.

EMBRYOLOGY

The esophagus is of double origin. The upper part, with the trachea being derived from the hinder part of the pharynx and the lower part arising from the primitive alimentary canal.

Up to the third week the trachea and the upper part of the esophagus form a single structure, the separation from the trachea occurring during the third or fourth week by the growth of two lateral septa which eventually meet and fuse in the midline to form a partition from which is differentiated the posterior (membranous) wall of the trachea and the anterior wall of the upper part of the esophagus.

During the second month the esophagus undergoes a rapid elongation due to the rapid increase in the size of the lung-buds.

Later, the esophageal lumen becomes narrowed by a proliferation of the epithelial lining. Some authorities (Kreuter) believe that the lumen becomes entirely obliterated while others (Forssner, Johnson) maintain that the original lumen is never entirely lost.

ETIOLOGY

It is now generally conceded that the anomaly is due to a developmental error arising at a very early stage, because in a 4 mm. long embryo the trachea is quite separate from the esophagus.

In the Harvard embryologic collection

* From the Department of Radiology of Gouverneur Hospital, New York City.

there is an 18.1 cm. long embryo showing this esophageal abnormality (Hirsch).

The developmental error for the type with the fistula occurs by the failure of meeting of the two lateral septa thus failing to differentiate the upper half of the esophagus from the trachea. The error for the type unaccompanied by the fistula occurs possibly as a result of a deviation in the line of development of the two lateral septa, or by a rupture of the continuity of the slender tube, which is the primitive esophagus below the tracheal bifurcation, by the rapid growth of the lung-buds.

SYMPTOMATOLOGY

The infants are often subjects of congenital asthenia together with other malformations. From the time of birth there seems to be an unusual amount of mucus constantly expelled from the mouth due to the fact that the oral secretions cannot pass into the stomach. Efforts at swallowing are attended by prompt regurgitation of the food mixed with the mucus through the mouth and nose. There may be violent suffocative attacks with severe cyanosis.

Fig. 1. Ante-mortem barium examination made on the fourth day after birth, demonstrating the upper dilated blind pouch of the esophagus ending at the level of the tracheal bifurcation.

Fig. 2. Post-mortem examination on the seventh day, demonstrating the extent of the lower pouch by the injection of barium into the stomach and exposing the film in the Trendelenburg position.

CASE REPORT

An infant, aged four days, weighing 6 pounds and apparently normal at birth, was referred to the roentgen department with a history of having regurgitated everything taken by mouth since birth. The vomitus was non-acid and contained frothy mucus. Only meconium was passed by rectum since birth.

Roentgen Examination. Barium sulphate was added to the baby's formula and the opening in the nipple enlarged. The mixture was taken eagerly. Roentgenoscopically, there was revealed a dilated blind cul-de-sac at the level of the tracheal bifurcation. No barium passed through this point. When the pouch became filled the baby vomited. No air was noted in the stomach.

Subsequent Course. A gastrostomy was performed under local anesthesia. The stomach was small. The lower blind pocket extended 1 inch up from the cardiac sphincture. Subsequently, the baby failed rapidly, dying from pneumonia on the seventh day.

Necropsy. There was found a congenital absence of the middle of the esophagus, unaccompanied by any tracheal fistula, connecting tissue or other gastrointestinal anomalies.

CONCLUSION

Congenital absence of the middle of the esophagus, unaccompanied by either a tracheal fistula, a fibromuscular connecting band, or by an associated anomaly is the rarest form and its infrequent occurrence prompted the presentation of the above case.

REFERENCES

1. Abel, A. L. Oesophageal Obstruction, Oxford University Press, New York, 1929.
2. Forssner. Quoted by Abel, ref. 1.
3. Hirsch, I. S. Congenital atresia of esophagus. *J. Am. M. Ass.*, 1921, *76*, 1491.
4. Johnson. Quoted by Abel, ref. 1.
5. Kreuter. Quoted by Abel, ref. 1.
6. Martin. *Obs. d. sc. med. Marseille*, 1825.
7. McClellan, R. H., and Elterich, T. J. Atresia of esophagus, with tracheo-esophageal fistula. *Am. J. Dis. Child.*, 1923, *26*, 373–378.
8. McKenzie. Manual of Diseases of Throat and Nose, 1884.
9. Neff, F. C. Congenital absence of middle of esophagus. *Am. J. Dis. Child.*, 1921, *22*, 57.
10. Plass, E. D. Congenital atresia of esophagus with tracheo-esophageal fistula; associated with fused kidney. *Johns Hopkins Hosp. Rep.*, 1919, *18*, 259–286.
11. Reynolds, R. P., and Morrison, W. W. Congenital malformations of esophagus. *Am. J. Dis. Child.*, 1921, *21*, 339.
12. Thomas, W. The Anatomie of the Human Bodies Epitomized. Sixth edition. London, 1703, pp. 240–242.

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THE EFFECT OF ROENTGEN RAYS ON THE STOMACH IN RABBITS*

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A LARGE number of investigations is available concerning the effect of roentgen rays on the secretory functions of the stomach both in the human and in animals. Briefly, the results of these investigations may be summarized as follows. Small and moderate doses have little or no effect on the gastric secretion in animals, large doses cause a reduction both of the daily secretion and of the free hydrochloric acid, the total acidity and pepsin. Clinically, the effect is found to be substantially the same, but it is less pronounced and less constant.

The experimental histopathological investigations, to which we shall strictly confine ourselves, are not so numerous as those relating to the functional changes, but many important and interesting observations have nevertheless been made. In experimental investigations with animals the majority of authors have found distinct changes in the stomach after roentgen irradiation (Regaud, Nogier and Lacassagne, Ghilarducci, Szegö and Rother, Dawson, Tsukamoto, Wolfer, Engelstad). These changes consist first and foremost of hyperemia, hemorrhages, and degeneration of the epithelium, but some authors have also found ulcerations (Ghilarducci, Tsukamoto, Wolfer, Engelstad). The last two authors point out the great similarity between these ulcers and the peptic stomach ulcerations in human beings. Other authors, such as Miescher, were unable to demonstrate any definite changes after roentgen irradiation. Clinical experiences do not suggest any particularly great radiosensitivity on the part of the mucous membrane of the stomach (Case and Boldyreff, Ledoux-Lebard and Piot, Delherm and Beau). A more extensive literary survey will be found in a review by Desjardins in 1931, in a work by Schürch and Uehlinger (1935) and in an article by myself also published in 1935.

In the last-mentioned work I reported the results of a number of experimental investigations carried out with rabbits. The radiosensitivity of the mucous membrane of the stomach proved to be rather great, as great as, or even greater than, that of the skin. After irradiation changes, to some extent very considerable, occurred in the shape of degeneration of the mucous membrane and inflammation, while in a large number of cases there were also ulcerations, not a few of which were perforating. These ulcerations of the stomach bore a striking likeness to the peptic ulcers in human beings, and they also had a corresponding localization.

These investigations on the effect of roentgen rays on the stomach have been continued, and it is the observations which have been made during their course which are to be reported on below.

The total material consists of 59 rabbits, all of which have been irradiated over a field in the epigastrium corresponding to the stomach. The physical conditions of the irradiation were as follows: tension 175 kv., strength of current 4 ma., filter 0.5 mm. Cu+1 mm. Al, focus-skin distance 27 cm., size of field 3×4 cm. to 4×6 cm., radiation intensity 41 r per minute. The doses administered varied considerably, as the tables show. In the majority of cases the entire dose was administered at one séance, but in some cases it was broken up and administered in 2–4 séances at intervals of up to one month.

1. Since the result of my previous investigations suggests that the radiosensitivity of the mucous membrane in the rabbit is very great I have endeavored to determine the minimum dose which induces changes in the mucous membrane of the stomach. The results of this series of experiments are given in Table 1.

* From The Norwegian Radium Hospital, Oslo (Chief: Rolf Bull Engelstad, M.D.).

Table I

Rabbit No.	Dose r	Duration of Life after Irradiation	Macroscopical Changes	Microscopical Changes
163	125	18 days	o	o
162	250	18 days	o	Slight hyperemia
157	500	28 days	o	Slight hyperemia, very slight degenerative changes in the peptic cells
141	500	30 days	o	Same as rabbit No. 157
142	1,000	22 days	o	Moderate degenerative changes, most pronounced in the peptic cells and the cells of the pyloric glands. Hyperemia
156	1,000	28 days	o	Substantially the same as in rabbit No. 142

It follows from these investigations that after a dose of 500 r very slight, but nevertheless distinct, changes are found in the peptic cells. In the other cellular elements of the gastric mucous membrane no certain signs of degeneration can be demonstrated. After the small doses there are no definitely demonstrable changes, inasmuch as the hyperemia which was observed after 250 r is so little pronounced that it does not permit any definite conclusions to be drawn, particularly as there are no other demonstrable changes. In view of this it can undoubtedly be taken for granted that the smallest dose which causes distinct changes lies between 250 and 500 r. After doses of 1,000 r the degeneration is still only moderate, although considerably more pronounced than after 500 r.

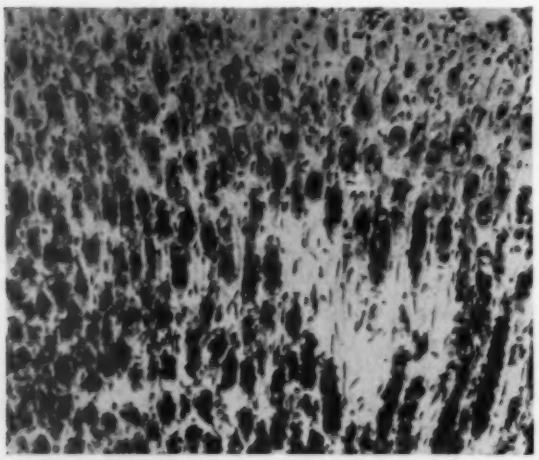

Fig. 1. Degenerative changes in the peptic cells (225×).

This series of experiments also shows that it is the peptic cells of the stomach glands which are most radiosensitive (Fig. 1), a circumstance which has previously been pointed out by Regaud, Nogier and Lacassagne.

2. In the next series of experiments which are to be referred to, 10 rabbits have been given, in one séance, a dose of 1,500 r. The results will be found in Table II. This table contains only a very brief and schematic description of the findings which will be more specifically referred to below.

Undoubtedly, a dose of 1,500 r may seem to be very large, but viewed in conjunction with the skin tolerance of the rabbit it must, nevertheless, be said to be comparatively moderate. In previous works I have shown that it is necessary to administer a dose of about 1,700 r before a distinct skin reaction is obtained, and even considerably larger doses must be administered to call forth a pronounced exudative epidermitis. In my previous investigations, therefore, I have divided the doses administered into subepidermicidal, epidermicidal and dangerous or lethal doses. The experimental series referred to above shows that very considerable changes in the stomach can be caused as a result of a dose which is not only subepidermicidal but is even considerably smaller than the epidermicidal doses. In other words, this shows as plainly as could be desired that the stomach mucous membrane cannot be regarded in any manner as radioresistant, as a number of work-

Table II

Rabbit No.	Duration of Life after Irradiation	Macroscopical Changes	Microscopical Changes
171	1 day	o	Degeneration in the lymph follicles; hyperemia
191	3 days	o	o
170	7 days	Erosion on the lesser curvature of the corpus	Slight degeneration particularly of the peptic cells. Hyperemia. Slight leukocytic infiltration
169	15 days	Ulcer near cardia. Blood in stomach	Typical ulcer. Degeneration. Hyperemia. Leukocytic infiltration
166	16 days	Ulcer on the lesser curvature in the corpus	Same as rabbit No. 169
164	22 days	"Kissing ulcers" on the lesser curvature of the corpus	Same as rabbits No. 169 and 166
165	24 days	Perforating ulcer in the region of incisura angularis	Typical perforating ulcer. Extreme degeneration. Otherwise substantially as in rabbit No. 166
168	24 days	Two ulcers in the region of the cardia, the one almost perforating. Blood in the stomach	Typical ulcer which reaches right down to the subserosa. Otherwise substantially as above
153	28 days	Perforating ulcer near incisura angularis	As in rabbit No. 165, very pronounced changes
167	46 days	Perforating ulcer near the cardia	Substantially as the preceding

ers have asserted, but that, on the contrary, it must be said to be rather radiosensitive, obviously more sensitive than the skin. The changes which are to be found are degeneration, not only of the peptic cells but also of the other cellular elements of the mucous membrane, hyperemia and leukocytic infiltration.

As early as twenty-four hours after irradiation a very slight hyperemia is discerned, the significance of which, however, is difficult to determine. Furthermore, degeneration of the lymph follicles is seen, a process which we know commences in the hours immediately succeeding irradiation. It therefore appears as if there is also a mild initial reaction in the stomach, but this has already run its course three days after irradiation.

After a lapse of seven days from the time of irradiation, the main reaction is already in progress and at all the subsequent observations very pronounced changes are to be found. Moreover, in all these animals ulcerations are found in the stomach, after the lapse of seven

Fig. 2. Erosion in the cardia (15×).

Fig. 3. Perforating ulcer (9×).

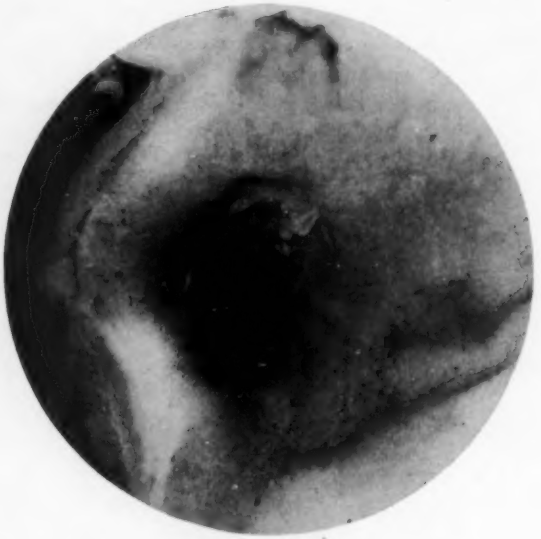

Fig. 4. Typical ulcer after roentgen irradiation (4×).

days in the shape of a small erosion only (Fig. 2), but subsequently as more or less deep, in some cases even perforating, ulcers (Fig. 3). It will be seen from the table that perforating ulcers were found in no less than 3 animals, all of which died of peritonitis. In 2 of the other animals such considerable quantities of blood were found in the stomach that it must be assumed it is this hemorrhage from the ulcers which was the cause of death.

The ulcers have the same typical appearance which I have described in previous works, where I have pointed out the striking similarity

Table III

Rabbit No.	Dose	Duration of Life after Irradiation	Macroscopical Changes	Microscopical Changes
201E	2,500 r	14 days	One ulcer near cardia and one near pylorus	Degeneration. Hyperemia. Slight leukocytic infiltration. Superficial ulcers
124	2,000 r	19 days	Ulcer near pylorus	Extreme degeneration. Inconsiderable leukocytic infiltration
123	2,000 r	19 days	Ulcer near pylorus	As in rabbit No. 124
137	2,000 r	25 days	Ulcer in canalis pylori on the posterior wall. Closed perforation	Considerable degeneration, hyperemia and leukocytic infiltration. Typical perforating ulcer
181	2,200 r	28 days	Ulcer on the lesser curvature	As in rabbit No. 124
143	2×1,000 r	38(8) days	Ulcer near incisura angularis	Typical ulcer. Otherwise as in rabbit No. 201 E
140	2×1,000 r	46 (15) days	Ulcer near cardia. Blood in stomach	As in rabbit No. 124
146	4× 500 r	7 (4) months	Small scar	Small scar. Otherwise nothing noticeable

to the peptic ulcers in human beings. The roentgen-ray ulcers in the rabbit are round or oval, not rarely crater shaped, sharply defined and surrounded by more or less wall-shaped borders (Fig. 4). The size may vary somewhat, but not greatly; as a general rule, the fully developed ulcers are about 1 cm. in diameter. The floor of the ulcer is often covered with a grayish-white adhesive layer.

3. In a third series, somewhat larger doses have been administered, from 2,000 to 2,500 r, in 1 up to 4 séances. The intervals between the individual séances have always been one month. The results will be found in Table III.

In this series ulcers have been demonstrated, one of which was perforating, in all animals except the one which was given its dose most fractionated and which was not killed until seven months after the first irradiation and four months after the last. In this case there was probably also an ulcer which, at the death of the animal, was healed, but the changes were so little pronounced that it was difficult to say if this had been the case. In all animals in which, on post-mortem examination, ulcers were found in the stomach there was also pronounced degeneration of the mucous membrane, hyperemia and leukocytic infiltration which, in the majority of the cases, were moderate, but in one single case very marked.

After the administration of doses of this size there has been a very high mortality under the experimental conditions here used. No less than 6 of these 8 animals died during the course of the weeks immediately succeeding the irradiation.

4. As a result of a dose of 3,000 r administered at 1 séance the mortality is even greater. Of 13 animals which received this dose 1 was killed five days after irradiation, in other words before the full effect could be expected, all the others died during the course of the six to seven weeks immediately succeeding the irradiation; and there were only 2 animals which lived more than three weeks after irradiation. The results of this experimental series are given in Table IV.

In the rabbit which was killed five days after irradiation no definite changes were found in the gastric mucous membrane, either macroscopically or microscopically. In the 2 animals which died seven and eight days after irradiation there was distinct degeneration, especially of the peptic cells. No definite ulcer was found; in both animals there was merely a slight erosion. In all the other animals very considerable changes were demonstrated in the wall of the stomach, and typical ulcers, 2 of which were perforating.

This experimental series thus shows that a dose of 3,000 r administered at one séance calls forth a very strong reaction in the wall of the stomach. The degenerative changes are found almost exclusively in the mucous membrane. In the deeper layers there is either no degeneration, or it is very little pronounced, apart from the narrow necrotic zone which surrounds the ulcers. The hyperemia attacks both the submucous coat, the muscular coat and the subserosa, but is always most pronounced in the submucous coat. After these doses the stomach wall is also the site of an edema which, particularly in the submucous coat, can be very pronounced. Leukocytic infiltration can be found in all layers of the stomach wall, especially when there are very deep or perforating ulcers, but it is always greatest under the mucous membrane and between the glands of the latter.

As far as can be determined, a dose such as that given in the present case will always cause stomach ulcerations, with an appearance which corresponds entirely to that of the peptic gastric ulcers in human beings, always assuming that the animal lives for a sufficiently long period after irradiation. During the first three or four weeks these ulcers show no sign whatever of healing, but in an animal which first came under post-mortem examination forty-six days after irradiation there was found a noticeable connective tissue formation under and around the ulcer, showing that there are reparative processes at work.

What, therefore, is the cause of the death of all these animals in the weeks immediately succeeding the irradiation? In 2 cases perforating gastric ulcers have been found, with diffuse peritonitis, and in 3 cases a considerable hemorrhage in the stomach, which is sufficient explanation. In the other cases, the cause must be looked for partly in the general intoxication which is a result of the irradiation, and partly in the regularly occurring inanition. It is always observed that some time after the irradiation the animals eat less and in the days immediately preceding death they take practically no food. This, undoubtedly, is due, in turn, partly to the fact that the general reaction produces a great anorexia and partly because it is painful for the animal to feed on account of the local reaction.

Table IV

Rabbit No.	Duration of Life after Irradiation	Macroscopical Changes	Microscopical Changes
110	5 days	o	o
122	7 days	Erosion	Degeneration, especially of the peptic cells. Inconsiderable leukocytic infiltration
121	8 days	Erosion	As in rabbit No. 122, but also degeneration in the pyloric glands
154	15 days	Ulcer near the pylorus	Considerable degeneration and hyperemia. Moderate leukocytic infiltration
155	17 days	Perforating ulcer near lesser curvature in the canalis pylori	Typical perforating ulcer with very pronounced changes
149	17 days	Ulcer near incisura angularis; hemorrhage	As in rabbit No. 154
150	18 days	Ulcer near incisura angularis; hemorrhage	As in rabbit No. 154, but somewhat greater leukocytic infiltration
152	18 days	Ulcer on the lesser curvature of the corpus; hemorrhage	Very great degeneration. Otherwise as in rabbit No. 154
158	18 days	Ulcer on the lesser curvature in the canalis pylori	As in rabbit No. 154.
159	19 days	Ulcer near the cardia, on point of perforation	Very great degeneration; moderate leukocytic infiltration
161	19 days	Ulcer on lesser curvature of the corpus	As in rabbit No. 154
160	25 days	Perforating ulcer on the anterior wall in the corpus near lesser curvature	As in rabbit No. 159
151	46 days	"Kissing ulcers" on lesser curvature of the corpus. Healing?	Pronounced fibrous changes under and around the ulcer. Considerable degeneration and leukocytic infiltration

Viewed in relation to the skin tolerance of the rabbit, 3,000 r is a very large dose which regularly causes epilation and a distinct epidermitis with scaling. The scaling, however, according to my experience from previous investigations, as a rule only takes the form of a dry desquamation. It is only very rarely that after these doses there appears a really exudative epidermitis with complete epidermolysis over the whole of the irradiated field. In that case healing also takes place very rapidly.

5. In order to investigate the significance of the fractionating of the doses for the effects of roentgen rays on the stomach, doses of between 3,000 and 4,000 r distributed over 2 to 4 séances have been administered to 12 animals. The intervals between the individual séances have been three to five days in some cases, and one month in others.

After these irradiations the skin reaction has been quite mild, with epilation, which has not always been total, and moderate desquamation, considerably less pronounced than in the preceding group. It also proves that after the fractionated irradiation the mortality is much lower. Whereas, in the preceding group it was

Table V

Rabbit No.	Dose	Duration of Life after Irradiation	Macroscopical Changes	Microscopical Changes
147	3×1,000 r	65 (3) days	Ulcer in course of healing in immediate proximity to incisura angularis	Degeneration of the mucous membrane, to some extent also slight hyperplasia. Moderate hyperemia and leukocytic infiltration. New formation of connective tissue
134	2×1,700 r	7 (5) days	Ulcer near the cardia	Mild degeneration. Hyperemia and leukocytic infiltration
135	2×1,700 r	13 (11) days	Ulcer near the cardia	Considerable degeneration. Hyperemia. Slight leukocytic infiltration
125	2×1,700 r	17 (14) days	Deep crater-shaped ulcer on the minor curvature near pylorus	Great degeneration. Hyperemia. Inconsiderable leukocytic infiltration. Edema
126	2×1,700 r	17 (14) days	Ulcer near cardia, on point of perforation	As in rabbit No. 125
151	2×1,700 r	22 (19) days	Ulcer near cardia, on the point of perforation	Extreme degeneration. In the small vessels degenerative changes and thickening of the intima. Considerable leukocytic infiltration. Edema
133	2×1,700 r	26 (21) days	Deep ulcer on the anterior wall in the sinus region. On point of perforation into the small intestine	Enormous degeneration, some necrosis. Considerable leukocytic infiltration. Edema
130	2×1,700 r	35 (31) days	Thickened, pale region near cardia. No ulcer	Enormous degeneration of mucous membrane and leukocytic infiltration. Edema
138	2×2,000 r	33 (3) days	"Kissing ulcers" near cardia	Degeneration, hyperemia and slight leukocytic infiltration
136	2×2,000 r	52 (21) days	Perforating ulcer on lesser curvature of the corpus	As in rabbit No. 125
145	4×1,000 r	7 (4) months	Scar near cardia	Scar-like fibrous changes. Ulcer in process of healing
148	4×1,000 r	7 (4) months	Scar near cardia	Substantially as in rabbit No. 145

necessary to count upon a mortality of 100 per cent, in this group of 12 animals only 5 died after irradiation. In the case of some of the animals the general condition was, as a matter of fact, not visibly affected. The results of the macroscopical and microscopical examination of the stomach are given in Table v. Only in one of these animals (No. 130) can no ulcer or scar be demonstrated, but even in this case there is a very considerable degeneration of the mucous membrane and leukocytic infiltration and also pronounced edema. Right from the first irradiation this animal had eaten very little and it is possible that the fact that the stomach was spared played a certain rôle. In all the other animals in this experimental series, either

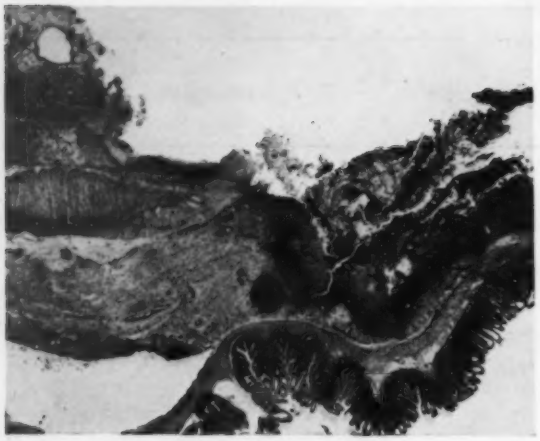

Fig. 5. Ulcer on the point of perforation to an adherent loop of the small intestine (12×).

ulcers or scars were found, and of the former, one was perforating and three or four on the point of perforation. In the one case the ulcer was on the point of perforation to an adherent loop of the small intestine (Fig. 5).

It is a striking fact that these ulcers arising from roentgen treatment are nearly always solitary. And in the very rare cases in which there are more than one, they are usually to be found as typical kissing ulcers, one on each side of the lesser curvature.

In the case of one of the animals in this experimental series, the distinct impression is gained that the ulcer is in process of healing (Fig. 6), and in 2 others there are scars or in any event ulcers which are practically healed. This is an observation which is of considerable interest, *inter alia*, because it can contribute to an understanding of the pathogenesis of these ulcers. This question will be discussed in more detail in a later chapter, but it may be remarked here that other workers have found that the gastric ulcers which appear in conjunction with irradiation show little or absolutely no tendency to healing. This has been emphasized by Schürch and Uehlinger in particular who carried out their irradiation by applying radium in the foramen of Winslow and thereby caused stomach ulcers which, in their appearance, very greatly resembled those which are mentioned in the present work.

6. In 3 animals a dose of 4,500 r has been administered at one séance, and 2 animals have been given the same dose spread over 3 séances at intervals of one month. All these 5 animals died from the irradiation eight to seventeen days after final administration. The results are given in Table VI. In this group there are gastric ulcerations in all animals, except one which died nine days after irradiation and where the possibility must be considered that the time was too short for the development of an ulcer.

If the findings in this group are compared with those in the preceding two, no noteworthy difference is seen in the degree of the changes. This may well be because the duration of the lives of the animals after irradiation is so short that there is not time for the development of even greater changes. Nor can there be said to be any essential difference here between the animals which were given the entire dose at one séance and those to which it was administered during the course of 3 séances at intervals of one month, probably because the individual doses are so large.

7. In Table VII have been set down the results of 5 experiments with injection of China ink into animals which had previously been treated by roentgen irradiation of the stomach. In this case intracardial injection of filtered undiluted Günther-Wagner's "China ink" was given. The thoracic cavity was opened, a cannula inserted in the left cardiac ventricle, and the China ink was allowed to enter through an irrigator held at a height of 1 to 1.5 meters. It has, however, proved extremely difficult to obtain a satisfactory filling with China ink in this manner, as the heart action fails before sufficient ink is pumped into the circulation. Attempts have therefore been made to give the injection in the vena femoralis after incision, but with no better result.

Fig. 6. Ulcer in process of healing. Epithelialization from the borders, fibrous changes (30×).

Table VI

Rabbit No.	Dose	Duration of Life after Irradiation	Macroscopical Changes	Microscopical Changes
114	4,500 r	9 days	No ulcer	Extreme degeneration; leukocytic infiltration; considerable submucous edema
117	4,500 r	13 days	Ulcer near cardia	Considerable degeneration; mild leukocytic infiltration
118	4,500 r	13 days	Ulcer near lesser curvature in the corpus	As in rabbit No. 117
139	3×1,500 r	69 (8) days	Ulcer near lesser curvature near the pylorus	Superficial ulcer. Considerable degeneration; much leukocytic infiltration; moderate edema
144	3×1,500 r	80 (17) days	Perforating ulcer on lesser curvature near the pylorus	Enormous degeneration; slight leukocytic infiltration; considerable edema; typical ulcer

It follows from Table VII that in one case (No. 200) an infarction-like region was found on the lesser curvature (Fig. 7). On the mucous membrane side there was found a sharply defined, quite pale region, with a diameter of not quite 1 cm. On the cut surface this proved to be triangular in shape with the apex turned towards the serosa. The borderline towards the China ink-filled surroundings was rather sharply defined. On microscopic examination no ink content was found in the blood vessels within the pale area. An examination of the blood vessels themselves, however, did not yield any information as to the nature of the vascular lesion which was the cause of the stoppage of the passage. The histological appearance of the vessels showed no deviations from the normal, nor in their content was it possible to demonstrate any certain pathological changes such as thrombosis, embolism or stasis. On the other hand, there was very considerable edema in this region.

Table VII

Rabbit No.	Dose	Duration of Life after Irradiation	Macroscopical and Microscopical Changes
199	2,200 r	14 days	Poor China ink filling. Superficial ulcer on lesser curvature in the corpus. Considerable mucous membrane degeneration. Moderate leukocytic infiltration
200	2,200 r	14 days	Good filling with China ink, infarction-like region on lesser curvature. Otherwise as in rabbit No. 199
202E	2,500 r	10 days	Poor filling with China ink. Ulcer on the lesser curvature near the cardia. Infarction? Otherwise as in rabbit No. 199
203E	2,500 r	7 days	Ulcer on the lesser curvature near the pylorus. Otherwise substantially as in rabbit No. 199
204E	2,500 r	11 days	Substantially as in rabbit No. 203E

In another of the animals (No. 202E) there was also a very small pale region but this did not have the characteristics of that first described, and as the China ink filling was less satisfactory here, this finding does not provide a certain clue to the presence of any vascular lesion. Nor, in this case, was it possible to demonstrate any obvious histological changes in the blood vessels.

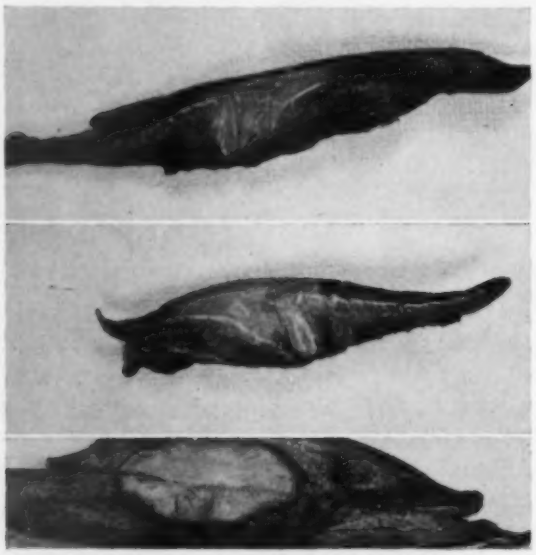

Fig. 7. Infarction-like region after China ink injection, seen from the mucous membrane side and from the cut surfaces (3×).

This experimental series does not permit any definite conclusions to be drawn. It can only be emphasized that in one single case an infarction-like region was found. It is, perhaps, not improbable that this characteristic picture may be due to a fortuitous circumstance in the distribution of the China ink in the blood vessels of the stomach, but the supposition cannot be excluded that it may be due to the existing edema. There is much which suggests that in this case there was a vascular lesion, but what its nature was, and whether it was primary or secondary in relation to the other changes it is impossible to state with any certainty.

DISCUSSION

It follows from the experiments which are here reported that roentgen irradiation of the stomach causes constant and characteristic changes, a reaction which shows complete agreement with what is found in other organs and tissues. This reaction consists of degeneration, hyperemia, in many cases increased transudation and leukocytic infiltration. Furthermore, there are to be found, even after comparatively moderate doses, gastric ulcerations which have a characteristic appearance and localization, and which are strikingly constant in their occurrence.

The degeneration is found first and foremost in the mucous membrane, in a much smaller degree in the other layers of the stomach wall. It can attack all the cellular elements of the mucous membrane, the surface epithelium, the peptic cells of the proper gastric glands, and the coating cells and cells of the pyloric glands. But there is a distinct difference in radiosensitivity, as the peptic cells of the gastric glands are considerably more radiosensitive than the other cellular elements of the mucous membrane, a fact which is pointed out by Regaud, Nogier and Lacassagne and to which I, also, have drawn attention in a previous work.

Here, as in other organs, the degenerative changes consist partly of swelling and vacuolization of the cells, partly of pyknosis and to some extent also of complete destruction and dissolution. There also occur, particularly in the rather later stages, more or less monstrous cellular forms. During healing, regeneration of the epithelium is found, and in some cases a somewhat more marked, but always moderate, epithelial proliferation. In no case has it been possible to demonstrate autonomous growth.

The degeneration in the other coats of the stomach wall, the submucosa, muscularis and serosa, is always considerably less pronounced and is to be found preferably on the floor and ridges of the ulcerations. Thus, unlike Schürch and Uehlinger, I have not been able to discover any specially pronounced radiosensitivity of the serosa and subserosa, but this may be due to the method of irradiation, as the authors in question applied radium, filtered with 0.5 mm. platinum and 1 mm. rubber, into the

foramen of Winslow. In these experiments the serosa has always been most closely situated to the source of irradiation and even if the radium tube were fitted into a rubber sound, the possibility of a certain caustic effect cannot be entirely excluded.

It has only been possible in a very few cases to demonstrate morphological changes in the blood vessels, and these have consisted in part of thrombosis, and in part of thickening of the intima in the small vessels. It has not been possible with the staining methods employed in these experiments to demonstrate any definite morphological changes in the sympathetic nerve cells in the stomach wall, but no special stains have been used. The stomach specimens have been fixed in Bouin's fluid and stained by Masson's three-color method.

The degenerative changes in the mucous membrane of the stomach are distinctly demonstrable seven days after irradiation. Before this period has elapsed there are no definite changes in the membrane itself, but even the first day a very pronounced degeneration is found in the lymph follicles of the stomach wall. As a result of previous experience with irradiation of this tissue, there is reason to believe that the degeneration begins here during the actual irradiation or, in any event, in the course of the two hours following the irradiation (Engelstad). The degeneration of the mucous membrane seems to reach its maximum during the first or second month after irradiation, but previous experiments show that a very long time must elapse before it completely recedes. In any event, as long as five months after irradiation it has been possible to demonstrate monstrosities in the epithelial cells.

In addition to the degenerative changes we find, as already mentioned, hyperemia, edema and leukocytic infiltration. The hyperemia can appear as early as one day after the irradiation, but is first found in a more pronounced degree after the lapse of a week. It is very possible that here, as in the same way with the skin, there is a brief initial reaction, which is then followed by a period of latency lasting a few days before the main reaction commences. The edema is of varying intensity and is usually to be found when there is also otherwise a strong reaction. It is therefore usually most pronounced when the other changes are at their height, from the second week onwards to the fifth and sixth after irradiation. The leukocytic infiltration, which commences after approximately one week, can also vary greatly. In some cases it is almost absent, in others exceptionally great. There are to be found partly granulocytes—usually in the early stages—partly lymphocytes, and the latter, in any event in the later stages, are absolutely dominant. In some cases a great abundance of cells is to be found with an appearance like macrophages. The cellular inflammatory reaction has its maximum at approximately the same time as the degeneration of the mucous membrane and subsequently recedes gradually, but it may be present to a great degree as late as five months after irradiation. From the second month it is not a rare occurrence to find a more or less pronounced new formation of connective tissue (Fig. 8).

Briefly, the following is the course of the reaction of the stomach wall after roentgen treatment: There is, in all probability, an initial reaction which commences during the course of the first twenty-four hours and which is very brief. This is followed by a period of latency of a few days' duration. About a week after irradiation the main reaction sets in and becomes continually greater during the immediately succeeding weeks. It reaches its maximum in the course of the first and second month, as a rule three to four weeks after irradiation. From the second to the third month the changes gradually decline but may still be in evidence five months or more after irradiation.

In previous publications I have pointed out that the stomach is not so radioresistant as some workers have assumed. Thus, I have shown that a dose of 1,700 r admin-

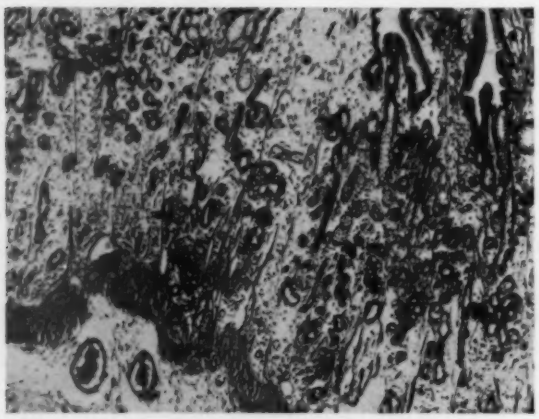

Fig. 8. Marked production of connective tissue following the reaction in the mucous membrane (90×).

istered at one séance, a dose which either evokes no skin reaction or only a very mild one, may be sufficient to produce a pronounced degeneration in the gastric mucous membrane, with development of ulcerations. I have pointed out that as a result of the investigations referred to, it must be justifiable to assume that the radiosensitivity of the stomach is just as great as, or even greater than, that of the skin. This view has been further strengthened by the experiments reported on in the present work. As is seen in Table 1, even after a dose of 1,000 r there are to be found undoubted, if moderate, changes in the gastric mucous membrane, and hyperemia. In not one of my cases has this dose caused any macroscopically demonstrable skin reaction. But also as a result of smaller doses there can be a demonstrable reaction in the wall of the stomach. The smallest dose which causes distinct changes seems to lie between 250 and 500 r.

In the discussion of the results of the experiments I have mentioned the rôle played by the fractionating of the radiation. There is no reason to go more closely into this here. I will merely emphasize the fact, well known from all radiobiological research work, that when the radiation is administered fractionally both the local and the general reaction are weaker than when the same dose is administered at one séance, and the strength of the reaction depends on the degree of the fractionating and the intervals between the individual séances.

ULCERATIONS OF THE STOMACH AFTER ROENTGEN IRRADIATION

It is seen from the tables that gastric ulcerations have been demonstrated in a large number of the experimental animals, so frequently that these ulcerations can be regarded as a characteristic feature in the reaction of the stomach to irradiation with roentgen rays, at least after the somewhat larger doses. It proves, namely, that among the 59 experimental animals there was demonstrated in 3 cases erosions, in 42 cases fully developed ulcers and in 3 cases scars. In other words, an ulcerating process has been demonstrated in 48 cases. Of the 42 ulcers, 8 were perforating and 6 on the point of perforation. There were only 11 animals in which no ulcer could be demonstrated, but in the majority of these cases the reason is very easily explained. To some extent the doses were too small to cause an ulcer, and to some extent the duration of life after irradiation was too short to permit the ulcers to develop. Thus, this was the case with 6 animals which were given a dose of 125–1,000 r and with 3 animals which only lived one, three and five days after irradiation. One animal lived nine days after a dose of 4,500 r without any ulcer developing, in spite of the fact that enormous degenerative changes were found. It is not improbable that, in this case also, an ulcer would have developed if the animal had lived longer. Finally, there was one animal which received a dose of 2×1,700 r and which lived 35 (31) days after without showing an ulcer, although in view of the magnitude of the dose and the length of the period of observation, there was every reason to expect that one could develop. In this case only a thickened, pale, edematous region was found in the vicinity of the cardia and an enormous degeneration of the mucous membrane and leukocytic infiltration, but no macroscopically demonstrable ulcer.

Gastric ulcerations after roentgen treatment have an appearance which corresponds entirely with that which is found in peptic ulcers in human beings. This applies particularly to the ulcers which are examined two weeks or more after irradiation; these we find to be round or oval, sharply defined, frequently crater-shaped, surrounded by more or less wall-shaped borders. Their size varies somewhat, but not very much, and as a rule they have a diameter of about 1 cm. The floor of the ulcer is generally covered with a grayish-white adhesive coating with a necrotic appearance. If the ulcers are situated in the immediate vicinity of the pylorus, the pyloric ring is always considerably thickened. The ulcers which are examined during the first two weeks after irradiation have, in some cases, the same appearance as that just described, while some are more flat, shallower and on a level with the surrounding mucous membrane, which shows no thickening or only an inconsiderable amount. Here also the floor of the ulcer has, as a rule, a necrotic coating. These ulcers are also round and oval, with sharply defined, clear cut ridges.

On microscopical examination a pronounced degeneration of the mucous membrane is found around the ulcer (Fig. 9); the degenerative changes in the submucous coat, and particularly in the muscular coat and the serosa are confined, in the great majority of cases, to the areas immediately

Fig. 9. Typical ulcer after roentgen irradiation (9×).

Fig. 10. The degeneration in the deeper coats is limited to the borders of the ulcer (48×).

adjacent to the floor and ridges of the ulcer (Fig. 10). The floor of the ulcer presents otherwise the same construction as that described by Askanazy in the case of human gastric ulcers. In practically all cases the following layers can be separated: a necrotic layer, a layer of nuclear remnants ("Kerntrümmerschicht"), a fibroid layer and a granulation or infiltration layer. The leukocytic infiltration under and around the ulcer may vary in degree; in the majority of cases it is moderate, but in some it can be very heavy and in others almost lacking. In the later stages the lymphocytes are predominant in these infiltrates; in the earlier stages numerous granulocytes may also be found. Macrophages also occur. In 2 cases wholesale occurrences of such cells were seen. Around the old ulcers there is always a distinct new formation of connective tissue (Fig. 11) and they give the impression of tending towards scar-like healing and epithelialization (Fig. 6). The epithelium which covers the puckered parts is generally cylindrical or cubical, but very irregular cell formations are often found and not infrequently monstrosities. In my previous experiments I found, even as late as five months after irradiation, very considerable changes in the wall of the stomach, in the shape of

degenerative changes, leukocytic infiltration and new connective tissue formations.

In only a very few of the cases do the blood vessels in the vicinity of the ulcer show distinct morphological changes. On the other hand, the arrosion of vessels does not seem to be so very rare. Thus, in the case of 6 animals such large quantities of blood were found in the stomach at the post-mortem examination that this must be assumed to have been the cause of death.

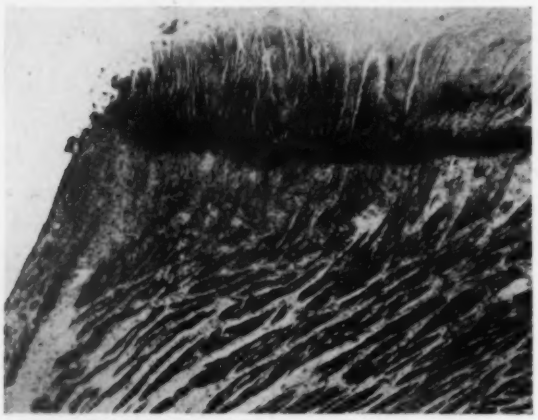

Fig. 11. Border of an ulcer. Extreme degeneration of the mucous membrane. Very marked fibrous changes (30×).

In earlier works I have attached great importance to the localization of the ulcers for the understanding of the pathogenesis. It proved that all ulcers were localized in the lesser curvature and its immediately adjacent surroundings, in other words, along the so-called "Magenstrasse." This circumstance comes up again with striking regularity also in these last series of experiments. All erosions, ulcers and scars are localized on the lesser curvature or immediately in front of or behind it. In the 48 ulcers or scars which have been found in these experiments the localizations were in other respects as follows: in the cardia in 14 cases, in the incisura angularis in 6 cases, near the pylorus or in the canalis pylori in 12, both in the cardia and the pylorus in 1, and otherwise in the stomach (i.e., practically speaking only in the corpus) in 15 cases. It cannot feasibly be assumed that this striking regularity in the localization of the ulcers to the lesser curvature rests upon pure chance. It is reasonable to suppose that it is due to anatomical and physiological characteristics of this region. This question will be more closely dealt with in the discussion of the pathogenesis.

In view of the fact that by the method employed a very large field of the stomach is irradiated, it is remarkable that more or larger ulcers are not found. As already mentioned, the size of the ulcers is very constant, with a diameter of about 1 cm., while the area which shows a distinct irradiation reaction is considerably greater, according to the size of the field employed. In other words, no ulceration is caused which occupies the whole of the irradiated area, nor do the ulcerations occur casually at any place whatever in this area. On the contrary, there are small, limited ulcers with a typical localization, in the great majority of cases solitary. In only 5 animals has more than one ulcer been demonstrated, and never more than two in any animal. In one of these there was one ulcer in the cardia and one in the pylorus. In the other cases, the two ulcers were typical "kissing ulcers" situated on each side of the lesser curvature.

The gastric ulcers which appear after roentgen treatment develop very rapidly. As I have already stated, no definite changes are found in the wall of the stomach in the week immediately following irradiation, apart from the mild initial reaction; nor, during this period, is there any ulcer to be found in the stomach. But simultaneously with the onset of the main reaction, about one week after irradiation, ulcers are regularly found, if the dose administered has been large enough. The earliest period for the occurrence of an ulcer is seven days after irradiation. In the course of the immediately succeeding two or three weeks the ulcer seems to develop very rapidly; in any event, in the animals examined post mortem during this period ulcerations are found in practically all

cases, and even two to three weeks after irradiation these ulcers have become very deep. Perforation, as a rule to the free peritoneal cavity, frequently occurs; of the 42 ulcers, 8 were perforating and 6 were on the point of perforation. The duration of time from irradiation until death in the case of the 8 animals with perforating ulcers was 24, 28, 46, 25, 17, 25, 52 (21) and 80 (17) days respectively. In the case of the animals with ulcers on the point of perforation the duration of life was 24, 19, 17, 17 (14), 22 (19) and 26 (21) days respectively. It proves, in other words, that perforation at the earliest has been observed seventeen days and at the latest forty-six days after irradiation. With the exception of the last mentioned case, all perforations have taken place in the course of the third and fourth weeks after irradiation (or after the final irradiation).

The reason that so many of these ulcers proceed to perforation is probably because there is not time for the development of a sufficiently protecting granulation and connective tissue, partly on account of the rapidity with which the ulcerations penetrate in depth and partly because, as present experience shows, no fibroblast proliferation begins until a month or more after roentgen irradiation. These two circumstances are, no doubt, two sides to the same question, as the rapid progression of the ulcers is not only dependent on the ulcer-inducing factors and their intensity, but also, in a high degree, on the deficient fibroblast proliferation. The fact that the perforation takes place so comparatively early, only one and a half to three weeks after the ulcerating process can be assumed to have commenced, is also a cause of the lack of formation of adhesions, for which reason the perforation generally takes place into the free peritoneal cavity and leads to a diffuse peritonitis. In only one case was a closed perforation found; in another the ulcer was on the point of perforation to an adherent loop of the small intestine. Otherwise no adhesions were found, or only few inconsiderable ones. It will be of great interest to acquire information as to the further development of the ulcers in the animals which do not die—of perforational peritonitis or some other cause—or are killed in the course of the first two months after irradiation. In the material presented here, there are only 3 animals with a longer duation of life. These animals were killed seven months after the first, four months after the last, irradiation. In all of them scars were found after gastric ulcerations. One of these animals had been given $4 \times 1{,}000$ r. In an animal which had been given $3 \times 1{,}000$ r and which lived sixty-five days after the first, three days after the last, irradiation, an ulcer was found which bore distinct marks of being in a process of healing.

Even though this material of protracted observations is small, and therefore permits of only limited conclusions, it shows, however, that there are possibilities of healing of roentgen ulcers in the stomach, as healing has taken place in all the 3 animals which lived four months after the final irradiation. My previous experiments have also shown that these ulcers can have a tendency to heal. In contrast to this Schürch and Uehlinger, who administered their radiation in the foramen of Winslow, have *never* been able to demonstrate any healing of the ulcers caused in this manner. Even in a case in which the gastric ulcer was 239 days old not the slightest sign of scar formation or epithelialization could be found. In only one case, that of a callous ulcer 114 days old, was it possible to demonstrate, histologically, a tendency to epithelialization.

In this work I have already demonstrated that the dose which is necessary to cause distinct histological changes in the mucous membrane of the stomach seems to lie between 250 and 500 r. In order to cause stomach ulcerations considerably larger doses are necessary. In no single case has an ulcer been demonstrated after 1,000 r or less. On the other hand, after doses of 1,500 r ulcers regularly appear. The minimum dose which can cause

roentgen ulcers in the stomach lies, therefore, between 1,000 and 1,500 r, with the irradiation technique used in these experiments. In Table II I have reported on an experimental series of 10 animals to which a dose of 1,500 r was administered. In all the cases in which the period of observation has been one week or more, in other words in the case of 8 animals, ulcers are demonstrated, no less than 3 of which were perforating and 1 at the point of perforation. In this series the frequency of perforation may seem to be strikingly high in comparison with the series in which larger doses have been administered, but the main explanation of this is, undoubtedly, that after the larger doses there are only few animals which live long enough to permit perforation because the mortality in the weeks immediately after irradiation is so much higher. But we must also take into account the fact that there may be great individual variations in radiosensitivity, especially when we are using a material, the composition of which is little homogeneous both in genetic respects and as regards conditions of life prior to admission to the laboratory. Moreover, complicating factors can arise which may be of essential importance. Thus, the stronger doses induce a stronger general reaction, which is assumed to be the main cause of the higher early mortality in these series, and which causes the animals to eat less during the period in which they are alive thus sparing the stomach mechanical and chemical irritations, a circumstance which may possibly be of great significance in relation to the development of the ulcers. It may be mentioned, in this connection, that in the one animal in which, 35 (31) days after a dose of 2×1,700 r, no ulcer could be demonstrated, the anorexia and aversion to food after irradiation was so great that even after the first administration it only ate very little.

But even if we must reckon with individual variations in radiosensitivity and complicating factors in the experiments, it nevertheless follows from my investigations that, after doses of 1,500 r and more, administered according to the method employed in these experiments, gastric ulcers can be caused in nearly 100 per cent of the cases.

It is difficult to form any definite opinion in regard to the *pathogenesis* of the gastric ulcers after irradiation with roentgen rays. The possibility of a vascular lesion as a reason for the development and maintenance of the ulcers is not inconceivable. In the case of the ordinary peptic ulcers in human beings, with which the roentgen ulcers in their macroscopic and microscopic appearance show a striking likeness, it has been emphasized by many workers, first and foremost by Hauser, that changes in the blood vessels more than anything else are the origin of the ulcers and their chronic course. It follows definitely from my material that pronounced functional changes in the blood vessels take place after roentgen irradiation, inasmuch as we regularly find in this material hyperemia and increased transudation. Morphological changes in the blood vessels, on the other hand, are only found in a small minority of the cases and here the other changes, degenerative and inflammatory, were simultaneously very great. Schürch and Uehlinger also find that in the stomachs irradiated with radium the changes in the blood vessels are strikingly small.

I have endeavored to obtain further light on the subject of the condition of the blood vessels by injecting China ink seven to fourteen days after irradiating the stomach with roentgen rays. I experimented on 5 rabbits, but it proved to be extremely difficult to inject sufficient China ink before the heart action stopped. For this reason the ink filling did not prove completely satisfactory in the majority of cases. In the case of one animal which was injected fourteen days after a dose of 2,200 r I was able to obtain a good filling and found on the lesser curvature an infarction-like, pale part, oval in shape on the mucous membrane side and practically triangular on the cut surface, with the top

pointed towards the serosa side. From the histological examination, however, it has not been possible to obtain any information in regard to the nature of the vascular lesion. No sign of thrombosis or stasis has been found. It has not, therefore, been proved if there is a real infarction, and even if it is regarded as one there is great doubt as to the significance which can be attached to it, whether it must be assumed that this process is the primary stage in the development of the ulcer, or if it is to be regarded as secondary in relation to the other changes. On the other hand, it is also possible that there is no infarction but that this appearance has arisen on account of the considerable edema which was found in the pale non-filled area. Taken into consideration with the fact that no corresponding feature was found in any of the other 4 animals, all of which had gastric ulcerations, and that in the whole of the material it was only quite exceptionally that morphological changes were demonstrated in the blood vessels or their content, this single case does not provide a sufficient basis for an assertion that the vascular lesions are commonly the primary stage in the development of the ulcer. The findings yielded by the material as a whole make it most probable that the vascular lesions are not the main cause of the ulcers and their further development.

The question is, thus, whether the stomach ulcers can be regarded as purely roentgen injuries, that is to say, as resulting wholly and solely from the irradiation, without other contributory causes, that, in other words they should be placed in the same category as the skin ulcerations which can occur after an overdose of roentgen rays. For several reasons this is very improbable. In the first place, it is difficult or impossible to believe that such serious roentgen-ray injuries can be sustained from such comparatively small doses as 1,500 r, a dose which, in my experiments, has regularly resulted in the development of stomach ulcerations, when we know that this dose does not cause any macroscopically visible skin reaction. And if the dose is measured on to the skin at 1,500 r, that which reaches the stomach at the lesser curvature, where the ulcers are always situated, is hardly greater than 1,000–1,200 r. It is proved that this is sufficient to cause pronounced degenerative changes in the mucous membrane of the stomach, but it is highly improbable that extensive lesions of the connective tissue and muscles would be caused by such a comparatively moderate dose. The investigations also show that no distinct degeneration is found in the submucous layer, the muscular layer, and the serosa, except in the immediate vicinity of the ulcers. If the entire process were simply a roentgen-ray effect one would expect to find signs of degeneration within the whole of the area irradiated, just as in the case of the mucous membrane. Furthermore, one would also expect to find ulcers which occupied the greater part of the irradiated area, or, in any event, in any part of this area. Instead, however, we find small, round, well-defined ulcers which are all localized on the lesser curvature or its immediate vicinity.

All these circumstances suggest that the effect of roentgen rays is not sufficient alone to cause these ulcers, even though it may be a necessary condition for them to develop. One or more other pathogenetic factors must also be present. In my opinion, therefore, the ulcers are not to be regarded as mere roentgen-ray injuries, but as acute or subacute stomach ulcers which, on account of the changes called forth by the irradiation with roentgen rays, have been given an opportunity of developing.

A similar point of view is advanced by Schürch and Uehlinger in connection with the radium ulcerations in the stomach. The lesion caused by irradiation with radium, which attacks the mucous membrane in particular, and most strongly its glandular parts, but which also in individual cases can attack all the coatings of the wall, results in the loss by the mucous membrane of the power to protect itself against self-

digestion, in such a manner that the wall coatings injured to the point of necrosis are dissolved by the gastric juice.

Even if it must be admitted in the case also of the roentgen ulcers that self-digestion may possibly play a rôle in the ulcer development, it is not probable, however, that it is the only factor, or the most essential, in addition to the radiation effect itself. In such case it would be impossible to explain why one therefore finds small, round, limited ulcers which only occupy a very small part of the area in which the mucous membrane is the seat of degeneration, and why there is always the same typical localization. I consider, therefore, that the self-digestion is hardly of any essential importance for the development of the ulcers, but that there must be other factors present which condition the characteristic appearance of the ulcers, their size and localization.

I have repeatedly emphasized on previous occasions that in their macroscopic and microscopic appearance the stomach ulcers arising after roentgen irradiation reveal an absolutely striking likeness to the ordinary peptic ulcers in human beings. This also applies to localization as all roentgen-ray ulcers are situated on, or in the immediate vicinity of, the lesser curvature, 14 near cardia, 6 near the incisura angularis, 12 near the pylorus, 1 near both the cardia and the pylorus and 15 on the corpus (and sinus). In the case of perforating ulcers and ulcers on the point of perforation, the localization was: 5 near the cardia, 2 near the incisura angularis, 4 near the pylorus and 3 in the corpus and sinus. The ulcers in human beings are also found mostly along the so-called "Magenstrasse" (v. Redvitz and Fuss). In Nicolaysen's material 52.5 per cent of the ulcers were found on the lesser curvature and 37.5 per cent in the pars pylorica. According to a large number of statistical figures compiled by Hauser, 79.19 per cent of the ulcers were situated on the lesser curvature, the posterior wall and pars pylorica, while 20.81 were on the anterior wall, the greater curvature, the cardia and fundus.

That gastric ulcers in human beings most frequently are localized along the lesser curvature is considered, on the basis of physiological and roentgenological studies of the passage of food through the stomach, and on the basis of experimental experience, to be due to the irritation which is set up by the passage of the food along the "Magenstrasse." The significance of these mechanical and chemical irritations has at an early stage been strongly emphasized by Stromeyer, Bloch and Nicolaysen and it now seems to be generally recognized that these factors play, in any event, a very important rôle. If all the characteristic qualities in gastric ulcers occurring after roentgen irradiation are taken into account, it is obvious that these factors make themselves strongly felt here also. I have also pointed out in previous works, that to my mind, the development proceeds in the following manner: The irradiation with roentgen rays causes a degeneration of the mucous membrane and a reduction of the vitality of the stomach. On account of this and of chance injury to the degenerated membrane caused by ingesta, erosions or small ulcerations, owing to the continually repeated irritation set up by the passage of the food, develop into larger, deeper and, as the case may be, perforating ulcers. If the animal does not die, of perforational peritonitis or from some other cause, the ulcers will subsequently, when the stomach has recovered its vitality and the regenerative processes have commenced, tend toward scar-like healing and epithelialization.

It is, naturally, not my intention with this opinion to assert that it is not possible to have gastric ulcers, and even perforating gastric ulcers, which are exclusively or mainly due to irradiation as such. If sufficiently heavy doses are administered it will be possible to cause a diffuse tissue necrosis, but the ulcers caused in this manner will, in all probability, have an entirely different appearance and a localization which is not so regular. Moreover, con-

siderably heavier doses would undoubtedly be required than those which, in these experiments, have resulted in ulcerations.

In addition to the mechanical and chemical irritants mentioned it may reasonably be supposed that there are other factors which may be of pathogenetic significance and which can, perhaps, also contribute to the characteristic appearance and localization of the ulcers. Mention has previously been made of the vascular lesions, which admittedly are rare, but which when they are present, may reasonably be thought to be of great significance. Similarly, reference has been made to autodigestion, which may also possibly play a certain rôle. But there are other factors which may have a bearing. It must be clearly borne in mind that when the rays are directed on the upper part of the abdomen it is not merely the stomach which is irradiated, but changes can also be caused in the liver, the spleen, pancreas, suprarenal glands, celiac ganglion, etc. What may be the effect of this, in the shape of changes in the internal secretion, changed functional condition in the vegetative nervous system, or in some other manner, is not yet known. It is also difficult or impossible to elucidate these questions as, under physiological conditions, an isolated irradiation of the individual organs cannot be effected.

The distinction which has been made above between the purely roentgen effect and the combined effect of roentgen irradiation and the mechanical-chemical irritation is, perhaps, first and foremost of theoretical interest and of importance in relation to radiobiology and the experimental ulcer research work. But it has also a certain amount of interest for practical therapy, as the consequence must be that in irradiations which affect the stomach the patient should be placed on a diet which is as little irritating as possible.

The question as to whether other conclusions of practical importance can be drawn from the observations which have been made here is one which is exceedingly difficult to answer. It is demonstrated that the stomach mucous membrane of the rabbit is very radiosensitive, and it would therefore be reasonable to expect also that malignant tumors arising from this membrane would prove to be radiosensitive. It has not, however, been possible to procure an experimental material for investigation in this field. Spontaneous stomach cancers, to judge by the literature, do not appear to occur in rabbits, nor have they ever been observed in any of the rabbits (about 250) which I have used for various investigations. Nor has an attempt to cause experimental stomach cancers by intramucous injection of 1-2-5-6-di-benz-anthracene led to any result. In 2 cases a marked proliferation of the mucous membrane around the point of injection has been demonstrated, but there has never been any sign of malignancy and in the majority of cases there has not been, on the whole, any demonstrable changes.

Even if one wished to count upon the existence of a certain parallel between the radiosensitivity of the different organs in the various kinds of animals, this could not also be applied as a matter of course to the stomach, because there are so many complicating factors here which may be of significance, *inter alia*, the type and composition of the food. Practical experience of human pathology does not suggest that the mucous membrane of the stomach possesses any especially high degree of sensitivity. Only very scattered reports have been received of serious stomach injuries in human beings after roentgen irradiation (Wetzel, Schönleber); in Halberstaedter's work of 1929 no case of roentgen injuries in the stomach is mentioned, and in Flaskamp's work of 1930 only the two just mentioned. Nor is the impression gained that the stomach cancers in human beings are susceptible in any noteworthy degree to radiation treatment. It is true that now and again after roentgen treatment a recession of the roentgenologically demonstrable changes is observed, but in these cases there is no means of knowing whether it is the tumor tissue itself which

is influenced or the accompanying infiltration of inflammatory nature. In any event, no complete destruction of the tumor is attained. Whether this is due to biological qualities in the tumor cells (radioresistance), unfavorable conditions in their milieu, or inadequate irradiation technique, it is not possible, at present, to express any definite opinion. But the results of the experimental investigations should, nevertheless, constitute a call for a continuation of the attempts to treat radiologically the stomach cancers which cannot be subjected to radical operation and also to give postoperative irradiation to those which can be operated on. At the same time the experimental work shows that great care must be exercised in irradiating the stomach, first and foremost, by protraction and fractionating of the radiation, non-irritating diet during the treatment and for a period thereafter, and careful attention to the patient, with frequent roentgen examinations.

SUMMARY

1. Irradiation of the stomach with roentgen rays has been carried out on 59 rabbits, with doses ranging from 125 r to 4,500 r.

2. After the heavy doses of 2,000 r or more the mortality is very high. But even after smaller doses (1,500 r) a number of the animals die.

3. After irradiation there is a mild initial reaction during the first twenty-four hours with degeneration of the lymph follicles and hyperemia, thereafter a period of latency, followed by the onset of the main reaction about seven days after irradiation, with degenerative and inflammatory changes.

4. The degeneration is found first and foremost in the mucous membrane, where, in turn, the peptic cells are most radiosensitive.

5. The other coatings in the wall of the stomach are only slightly radiosensitive.

6. In rabbits the radiosensitivity of the mucous membrane of the stomach is as great as, or greater than, that of the skin. Even after doses of 250–500 r there are histologically demonstrable changes and after doses of 1,000–1,500 r, which do not evoke any visible skin reaction, the changes are considerable.

7. If the radiation is fractionated, and thus also protracted, the changes are less pronounced and the mortality is reduced.

8. A dose of 1,500 r or more regularly causes ulcerations in the stomach with a macroscopic and microscopic appearance which shows an absolutely striking conformity to the peptic stomach ulcers in human beings.

9. The ulcers are always localized on the lesser curvature or its immediate vicinity (along the "Magenstrasse").

10. As a rule the ulcers are solitary, but "kissing ulcers" are also seen.

11. The ulcer development proceeds rapidly, in the course of the second to the fourth week after irradiation. Ulcers have been seen after seven days.

12. Perforation occurs very frequently, as a rule before the end of the first month, and generally to the free peritoneal cavity. The reason of the rapid progression and the frequent occurrence of perforation is to be sought, *inter alia*, in the lack of fibroblast proliferation during the first weeks after roentgen irradiation.

13. Considerable hemorrhage from the ulcers has been found in a number of cases.

14. If the animals live more than two to three months after irradiation the ulcers show a tendency toward healing and epithelialization.

15. The appearance and localization of the ulcers suggest that the pathogenesis is as follows: The roentgen lesion of the mucous membrane is a necessary condition for the occurrence of the ulcers, but together with this the mechanical-chemical irritation of the passage of the food is of great significance. Other factors can also play a part: vascular lesions, which however, can very rarely be demonstrated histologically (or by injection of China ink), autodigestion, and secondary effects

from other organs which have been irradiated simultaneously.

16. The results of the experimental investigations cannot, as a matter of course, be applied to the conditions in human beings, but they should, nevertheless, constitute a call for a continuation of the attempts to treat stomach cancers with irradiation, while at the same time they show that great care must be exercised in irradiating the stomach (protracting and fractionating of the radiation, non-irritating diet, careful control with frequent roentgen examinations).

REFERENCES

1. BLOCH, C. E. Beitrag zur Pathogenese des chronischen Magengeschwürs. *Deutsche med. Wchnschr.*, 1905, *31*, 1224–1228.
2. CASE, J. T., and BOLDYREFF, W. N. Influence of roentgen rays upon gastric secretion. AM. J. ROENTGENOL. & RAD. THERAPY, 1928, *19*, 61.
3. DAWSON, A. B. Histological changes in the gastric mucosa (Pavlov pouch) of the dog following irradiation. AM. J. ROENTGENOL. & RAD. THERAPY, 1925, *13*, 320–326.
4. DELHERM, L., and BEAU, H. Que faut-il attendre du traitement radiothérapique des tumeurs de l'estomac. *Paris méd.*, 1930, *1*, 190–192.
5. DESJARDINS, A. U. Action of roentgen rays and radium on gastrointestinal tract. AM. J. ROENTGENOL. & RAD. THERAPY, 1931, *26*, 145; 335; 493.
6. ENGELSTAD, R. B. Ueber die Wirkungen der Röntgenstrahlen auf die Lungen. *Acta radiol.*, Suppl. 19, 1934, pp. 1–94.
7. ENGELSTAD, R. B. Fourth International Congress of Radiology, 1934, Vol. *2*, 424.
8. ENGELSTAD, R. B. Ueber Magengeschwüre nach Röntgenbestrahlung. *Strahlentherapie*, 1935, *53*, 139–170.
9. ENGELSTAD, R. B. Ulcères de l'estomac chez le lapin après irradiation par les rayons roentgen. *Acta path. et microbiol. Scandinav.*, Suppl. 26, 1936, pp. 232–233.
10. FLASKAMP, W. Ueber Röntgenschäden und Schäden durch radioaktive Substanzen. (Sonderbände zur Strahlentherapie, Band XII.) Urban und Schwarzenberg, Berlin, 1930.
11. GHILARDUCCI, F. La radiogastrite ulcerosa nel coniglio, con speciale riguardo all'azione dei raggi secondari del bismuto. *Policlinico* (*sez. prat.*), 1916, *23*, 709–713.
12. HALBERSTAEDTER, L. Allgemeine biologische und schädigende Wirkungen der Röntgenstrahlen. In Jadassohn's Handbuch der Haut- und Geschlechtskrk., *5*, (2), Berlin, 1929.
13. HAUSER, G. In Henke und Lubarsch: Handbuch der spez. path. Anat. u. Histol., *4*, (1), Berlin, 1926.
14. LEDOUX-LEBARD, and PIOT, E. La radiothérapie pénétrante. Gauthier-Villars, Paris, 1930.
15. MIESCHER, G. Ueber den Einfluss der Röntgenstrahlen auf die Sekretion des Magens. *Strahlentherapie*, 1923, *15*, 252–272.
16. MIESCHER, G. Das Röntgenerythem. *Strahlentherapie*, 1924, *16*, 333–371.
17. NICOLAYSEN, K. Pathologisch-anatomische und experimentelle Studien über die Pathogenese des chronischen Magengeschwürs. *Deutsche Ztschr. f. Chir.*, 1921, *167*, 145–263.
18. VON REDWITZ, E., and FUSS, H. Die Pathogenese des peptischen Geschwürs des Magens und der oberen Darmabschnitte. Neue deutsche Chirurgie, Vol. *42*, Enke, Stuttgart, 1928.
19. REGAUD, C., and LACASSAGNE, A. *Radiophys. et radiothérapie*, 1927, *1*, 1.
20. REGAUD, C., NOGIER, T., and LACASSAGNE, H. Sur les effets redoutable des irradiations éntendues de l'abdomen et sur les lesions du tube digestif déterminées par les rayons de Röntgen. *Arch. d'électric. méd.*, 1912, *21*, 321–334.
21. REGAUD, C., NOGIER, T., and LACASSAGNE, A. Action nocive des rayons X sur le tube digestif. *Paris méd.*, 1911–1912, 489–491.
22. SCHÖNLEBER, W. Tod an Peritonitis nach Röntgenbestrahlung bei Peritonealkarzinose. *Strahlentherapie*, 1922, *13*, 126–128.
23. SCHÜRCH, O., and UEHLINGER, E. Strahlenveränderungen an abdominalen Organen. *Deutsche Ztschr. f. Chir.*, 1935, *245*, 261–278.
24. STROMEYER, F. Die Pathogenese des Ulcus ventriculi, zugleich ein Betrag zur Frage nach den Beziehungen zwischen Ulcus und Carcinom. *Beitr. z. path. Anat. u. z. allg. Path.*, 1912, *54*, 1–67.
25. SZEGÖ, E., and ROTHER, J. Ueber den Einfluss der Röntgenstrahlen auf die Magensaftsekretion. *Ztschr. f. d. ges. exper. Med.*, 1921, *24*, 270–288.
26. TSUKAMOTO, R. Ueber die Stoffwechselstörungen nach Bestrahlung der Leber mit Röntgenstrahlen. *Strahlentherapie*, 1924, *18*, 320–368.
27. WETZEL, E. Röntgenschädigungen mit und ohne Beteiligung der Haut. *Strahlentherapie*, 1921, *12*, 585–594.
28. WOLFER, J. A. Chronic ulcerations in the dog's stomach produced by x-ray. *Proc. Soc. Exper. Biol. & Med.*, 1925–1926, *23*, 45–47.
29. WOLFER, J. A. Chronic experimental ulcer of stomach; its clinical significance. *J. Am. M. Ass.*, 1926, *87*, 725–729.

RESULTS OBTAINED IN RADIATION TREATMENT OF INOPERABLE COLLUM CARCINOMA*

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THE cure of inoperable carcinoma forms the largest and at the same time the most important field of radiation therapy. Unfortunately the largest number of cases of carcinoma are found to be in such an advanced stage that operation can no longer be considered, leaving radiation therapy as a possible means of cure. Collum carcinoma, because of its tendency to rapid development, forms a high percentage of the inoperable cases, and consequently radiation therapy has here considerable importance.

Within a short period of time the cure of collum carcinoma has attained a great significance, and this frequently occurring form of cancer has from the first justified radiation therapy. The results obtained in this field have proved that it is possible, though only in a small percentage of cases, to cure collum carcinoma by radiation treatment, even when in an advanced stage. In a great measure it is due to the experience acquired in this field that radiation therapy is now used as a general treatment in the various forms of cancer.

If the tumor is in an advanced stage, we naturally cannot hope for a great deal of success, but considering the improbable possibility of an absolute cure, apart from the relatively small percentage of cures which may obtained in cases of inoperable collum carcinoma, even a temporary recovery lasting for a shorter or longer period of time, the so-called palliative effect, is of great importance.

At the Women's Clinic No. 1, Budapest, 702 cases of inoperable collum carcinoma were treated by irradiation during the ten years from 1919 to 1929. Thus, several years have elapsed since the last treatments, which were given in 1929. This period of observation, i.e., the many years that have elapsed, was necessary in order to evaluate the results obtained.

The malignancy of collum carcinoma is characterized by the swiftness with which it invades the neighboring tissues. The more important organs in the neighborhood—bladder, rectum, and ureter—are situated very near to this cancer and are connected to it by a net of lymphatic vessels. The parametrium surrounding the uterus offers an excellent field for the further spread. Winter stated, as is generally known, that collum carcinoma often reaches the limit of operability within four to six weeks, whereas corpus carcinoma remains in an operable condition for months. The manner and direction of the spread of collum carcinoma are various and totally incalculable.

The symptoms are blood-tinged discharge, hemorrhages, and pain. The last mentioned appears in the later stage. Those afflicted generally ignore the first two symptoms, and apply for treatment only when the last one appears. The pain usually signifies that the cancerous growth has extended into the neighboring tissues, chiefly into the parametrium, which in the majority of cases means an advanced inoperable condition. There are, therefore, two chief causes of this condition to account for the fact that the majority of cases applying for treatment are inoperable.

The great number of cases of inoperable collum carcinoma and the difficulties of its treatment are the explanation for the many experiments for its cure. We would have to go back many years if we were to sketch a perfect picture of attempts made to obtain a successful cure for collum carcinoma. In addition to the various caustic methods, attempts have been made to

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obtain results by chemotherapy, immunization, protein and serum therapy. Numerous other experiments have proved unsuccessful, with the exception of treatment by radium and roentgen rays. This treatment has proved successful to some extent. The numerous failures in the cure of this disease have been due to the fact that the vital energy of the cancer is so great that no cell or protoplasm toxin could possibly destroy it. According to our present knowledge only radium and roentgen rays are capable of destroying the cancerous growth and of retarding its development. The rays must exercise a restraining effect for a long period of time, because a destruction of the power of multiplication can only be obtained by the continuous influence of irradiation. Even if the influence of irradiation is not continued long enough, a transitory recovery sets in, the tumor regresses, but after a certain period of time is renewed. The difficulty of curing cancer is chiefly due to the fact that only a single cancerous cell or even only a fragment of it is necessary for regeneration.

The cancerous cells sensitive to the influence of radiation can be retarded in development or even destroyed by a suitable dose of radiation. As discovered from numerous experiences within this field, the treatment of collum carcinoma may be most efficiently rendered by a combination of radium and roentgen rays. The radium, which is employed for local treatment, has a more immediate and intensive effect, which however extends over a relatively small territory. For the treatment of the neighboring tissues, roentgen irradiation is used. In this manner, the action of radium is supplemented by roentgen rays and thus a uniform irradiation of the field is insured. The great obstacle to radiation treatment of collum carcinoma is the sensitivity of the adnexa to the rays.

Local radium treatment of collum carcinoma may be obtained by a combination of vaginal and cervical methods, insuring the cancerous area a thorough exposure. Where the cancer prevents the placing of radium in the cervical canal, no placement is to be forced which would destroy the tissues. It is advisable to treat the cancerous area first by means of radium; this should be applied in the vagina, and after three or four weeks, the treatment with radium is completed by the cervical method. In the vaginal method, the radium tubes used must have a heavy filter corresponding to 1 to 2 mm. of platinum. Caution should be used because the radium tube must be at least 1 cm. away from both the urinary bladder and the rectum. Even in a prone position it can easily happen that the radium tubes are shifted or moved, which endangers either the bladder or the rectum. Many attempts have been made to insure a successful radium treatment in collum carcinoma. Among the various methods used to fix the radium tube in the vagina, at the same time maintaining the proper distance from the bladder and the rectum, the best results have been obtained by employing different sizes and variously shaped cork containers for the radium tubes and ring pessaries of rubber construction. The walls of these insure the proper distance from the bladder, the rectum and at the same time act as a secondary filter. Concerning the employment of radium in cases of craterous decay of the uterine neck, the conditions are still more difficult. In such cases the great danger exists of radium overdosage, and possibly damage to the bladder and the rectum. In order to avoid this, it is necessary to determine the proper fixation of the radium tubes and the proper dosage of radium for the particular case in question. If the radium is to be used in the intracervical manner, it is advantageous to carefully dilate the cervical canal in order to avoid the destruction of the tissues which would bring about the danger of infection. In these cases we introduce radium tubes provided with a filter corresponding to 1 mm. of platinum in sterilized containers.

In regard to dosages for the treatment of inoperable collum carcinoma, the proper amounts to be used cannot very easily be

determined, because the effect of the radium rays depends on the quantity, distance, and the arrangement, and the filter strength. Moreover the degree of development of the tumor and the constitution of the patient are factors in the determination of the dosage; 4,000–6,000 mg-hr. of radium is the usual dosage. If, however, the vaginal method alone is employed, we must be satisfied with smaller dosages. Personal experience and the case in question determine the dosage. Extreme caution and sound judgment are absolutely necessary in the treatment of collum carcinoma in the advanced stages, because the tumor in these cases has extended to the fornix and the vaginal wall and therefore the bladder and the rectum are greatly endangered. The treatment of these particular cases should not be attempted by practicing physicians, but by radiologists of long experience in this field.

As a result of the irradiation, a gradual improvement sets in, and at the same time the tumor undergoes certain changes. The early effect of the radiation treatment consists in the softening of the cancerous tissues, which is caused by hyperemia as a result of edema. Two or three weeks after treatment, the tumor usually becomes smaller and later disappears. In case of the ulcerated tumor, the wound slowly becomes epitheliated, and after one or two months the speculum enables us to see a part of the portio, which in comparison to the decay is smaller or larger and which seems to be covered with healthy tissue. With the process of local healing, the disagreeable accompanying symptoms, chiefly the hemorrhages and the discharge—also improve. The effect of the irradiation decreases the virulence of the pus-forming bacteria; thus the cause of the odorous discharge is eliminated. In the change the discharge becomes mucus and loses its disagreeable odor.

The radiation treatment usually decreases the pain. The cause of this alleviation of pain can be explained in different ways. Probably one of the reasons is the cessation of pus formation. The degenerating tumor produces toxins which are absorbed into the system producing an irritative effect of the nerve endings. In our experience the hemorrhages showed a marked improvement after radiation treatment. In 80 per cent of the cases the hemorrhages ceased entirely. Somewhat later the amount of the discharge decreased, mainly 65 per cent. The least improvement was shown in the alleviation of the pain, namely in 30 per cent cessation and 50 per cent a decrease of pain. With radiation therapy an increased secretion of mucus is noticed and an accompanying decrease of pain. Coupled with this the hyperemia which follows irradiation beneficially influences the inflammation seen in tumors. As the tumors become smaller and disappear, the nerve endings are released from the pressure to which they have been subjected, which may explain the palliative results, though others make the irradiation directly responsible for this effect, stating that the rays have a direct action on the nerve endings. With the absence of the unpleasant symptoms accompanying the growths, anemia and the general condition improve, the system is capable of increased nourishment, the patients gain in weight and are able to resume their vocations. Thus, in most cases we can obtain a temporary improvement to postpone the inevitable end and render the interim bearable.

The retrogression and the disappearance of the primary tumor with its disagreeable symptoms does not necessarily mean that this pathological process is healed, for in the surrounding tissues tumor cells, undetectable by present-day methods, are latent and sooner or later cause a recurrence. Thus local treatment brings about only passing improvement and to achieve more enduring results we must endeavor to strengthen the system by means of plentiful nourishment and quiet life.

At the Women's Clinic No. 1 we have treated within ten years, from 1919 to

1929, 702 cases of inoperable collum carcinoma by irradiation. The systematic control examination was supplemented by the data received in letters with which we completed our post-treatment observations. We concluded the gathering of data on January 1, 1932.

From the therapeutic point of view, we can classify our material into two groups. The first group includes the palliative results, the second the successful cures. Not all of the 702 cases of inoperable collum carcinoma can be classified into the two above-mentioned groups because for a complete understanding, we must ignore the therapeutic viewpoint and simply divide the 702 cases into Group III, which includes 658 cases, and the remaining 44 into Group IV. Group III includes in a strict sense of the word an inoperable condition where the uterus is fixed and the carcinoma has invaded the neighboring tissues. Group IV is comprised of highly advanced and hopeless cases of inoperable collum carcinoma. From the histological point of view, we found carcinoma solidum in the greatest number of cases, and adenocarcinoma was rare. Our present knowledge has not been able to show a direct relation between the histological picture and the radiation treatment.

According to Forssell the absence of symptoms for one year after the completion of treatment shows a palliative effect directly resulting from radiation therapy. This he calls primary healing. From the standpoint of palliative effect, our material shows that of the 702 cases of inoperable collum carcinoma, 305 of the cases, or 43.1 per cent, showed primary healing.

In detail, the symptoms were absent for

1 year	155 cases
2 years	68 cases
3 years	23 cases
4 years	21 cases
5 years and over	38 cases

From the standpoint of complete cure, using as a basis at least five years with an absence of symptoms, we have had under our care 405 cases of the 702 for a period of at least five years after the primary healing. Of the 405 cases under our observation for five years, 38, or 9.3 per cent, were completely cured. In detail the time for cure:

5 years	14 cases
6 years	9 cases
7 years	6 cases
8 years	3 cases
9 years	1 case
10 years	1 case
11 years	2 cases
12 years	2 cases

Of the 44 hopeless cases belonging to Group IV, 18 lived for one year as a result of smaller passing improvement.

If we, for an easier survey, neglect the placing of inoperable collum carcinoma into groups referring to a duration of time, we reached immunity from symptoms for at least one year in 43.1 per cent of the cases out of the 702. Of the 405 cases observed for five years, 9.3 per cent showed a complete cure.

Our usual method of treatment of inoperable collum carcinoma is the following: We employ tubes containing 25 mg. of radium being supplied with a primary silver-brass-lead filter, which corresponds to a 1.5 mm. platinum filter in the vaginal method with a rubber ring, cork serving as a radium tube container, and at the same time acting as a secondary filter. Usually we use 50 mg. of radium; the time of irradiation extends from twenty-four to forty-eight hours. If at all possible we complete the vaginal radium treatment with the intracervical method. For this purpose we introduce into the cervical canal, after carefully dilating it, radium tubes with sterilized containers which are supplied with filters, corresponding to a total of 1 mm. platinum. With the combination of the vaginal and cervical methods 4,000 to 6,000 mg-hr. is generally used. We distribute the treatment over time intervals of several weeks. In the advanced cases of collum carcinoma where the cervix cannot be reached without

causing damage to the tissue, we only employ treatment by the vaginal method, and consequently use smaller doses of radiation than are employed in the combined method.

These treatments vary with the individual. Despite the fact that we have treated so many patients with the necessity of determining the proper dosage for each individual, we have had only 2 cases in which the deaths could be directly or indirectly connected with the radiation treatment. The following are brief reports of these 2 cases:

B. T., aged thirty-six, was admitted to the clinic on June 4, 1924, with a diagnosis of inoperable collum carcinoma. The disease was in an advanced stage. In the place of the destroyed portion there was an irregular, bloody crater. The right and posterior parametrium were deeply infiltrated. On June 6, after preparing the patient for treatment, we introduced 25 mg. of radium for 42 hours. Thereafter we dismissed the patient from the clinic and sent her home. Later we learned that three days after arrival at home she died suddenly. There was no autopsy.

F. J., aged sixty-five, was admitted to the clinic November 10, 1925, suffering from inoperable collum carcinoma. The disease had extended into the posterior fornix, and the left parametrium was intensely infiltrated. On November 12 excochleation took place. Then 50 mg. of radium was introduced into the crater. On the following day death occurred with symptoms of peritonitis. The autopsy report stated peritonitis diffusa and myodegeneratio cordis as the cause of death.

The first of these fatal cases took place suddenly at the home of the patient after she walked out of the clinic three days before. The death cannot be conclusively attributed to the radium treatment. In the second case, the excochleation played a more significant part in the peritonitis than did the introduction of the radium into the crater. Even though we attribute the causes of these two deaths to the treatment by radium, it shows only 2 fatal cases out of the 702, or 0.2 per cent. In 32 cases greater elevation of temperature and inflammation occurred; thus we can say the morbidity was 32 out of the 702 cases, or 4.5 per cent. After removal of the radium, however, these disturbances ceased within a relatively short time.

Injuries to the bladder seldom result from the use of radium. The edema of the bladder base noted during radium treatment disappears shortly after the treatment is completed. Complaints of injury to the rectum are more common than to the bladder. Even average doses of radium often cause tenesmus, which considerably influences the general condition; however, it usually disappears after a few weeks. If the cancer extends to the posterior or anterior vaginal wall, even our present-day technique does not render it entirely possible to avoid the formation of fistulas.

For external irradiation we use roentgen rays, at three times 3–6 fields, altogether 1,500 r per field. (Apparatus: radiotransverter, Coolidge tube, 190 kv., 5 ma., filter of 0.5 mm. Zn plus 0.5 mm. Al, skin-target distance 30 cm.) In the beginning the patients who were being irradiated were examined every sixth week, then every third month, later every six months. We were informed by letter, completing our observations, of those patients we could not keep in our care.

From the above it can be readily seen that in the treatment of collum carcinoma it is practically impossible to do without radium or roentgen rays. If we take into consideration the fate awaiting those afflicted with collum carcinoma prior to the present-day treatment, the value of our results can be seen. We can postpone the inevitable ending and at the same time make life in the interim more bearable.

To this, if we add the 8 to 10 per cent complete cures which may be obtained, even in such advanced cases, to the palliative effect it emphasizes the advantages of radiation therapy in the treatment of cancer.

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FACTORS INFLUENCING THE QUANTITATIVE MEASUREMENT OF THE ROENTGEN-RAY ABSORPTION OF TOOTH SLABS*†

X. TISSUE FACTORS

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IN CONSIDERING the factors which might influence the reproducibility of the I. Vs., especially those factors which are properties of the tissues being measured, we consider as important (*a*) the mass absorption coefficients, (*b*) the unavoidable slight surface drying during roentgenography, (*c*) the variation in thickness permitted without increasing the method error, and (*d*) the reproducibility of enamel and dentine I. Vs. as compared to similar calculations on known homogeneous filters of comparable absorption properties. The mass absorption coefficients and their variation with wave length, etc., are discussed at length in Paper IV. That the calculations made in Paper IV are substantially correct is shown by the experimentally determined D-T curves for enamel and dentine compared to aluminum given below.

A. WATER CONTENT OF DENTINE *vs.* I. VS.

From our first experiments, it was evident that the tooth slabs dried out a little on the surface while the roentgenograms were being made. However, since the drying may be supposed to occur only on the surface and since hydrogen and oxygen are of small atomic weight compared to the Ca and P of tooth substances, the removal of water should have little effect on the total absorption of the slab. We were thus inclined to regard the drying as a negligible factor. In order to show experimentally that it is negligible, we tested this possibility upon 4 tooth slabs, i.e., tooth No. 27 of thickness 0.86 mm.; tooth No. 40, 0.83 mm. thick; tooth No. 59, 0.82 mm. thick; and tooth No. 133, 0.95 mm. thick. Roentgenograms were made in the usual manner. Following this, most of the enamel was removed by drilling holes near the dentino-enamel-junction and chipping off the bulk of the enamel with a chisel. The dentine remaining was then weighed, dried for three days in an oven at 98° C., reweighed, and roentgenograms made again for comparison with the undried dentine.

In Table CXV are given the average D. Rs. in the various steps of penetrometers I and II in 2 films taken with moist tooth slabs at 30 kv., 30 ma., 15 inches, 0.85 sec., no filter, and black-paper-wrapped film. The D-T curves (not shown) are typical. The I. Vs. for the various dentine areas in the 4 teeth have about the usual variation between coronal and tooth dentine and may be described also as typical. From Figure 133, the appearances of the moist slabs (*A*) may be compared to that of the dry slabs (*B*) with the enamel chipped off. The drill marks are plainly evident (*B*). In Figures 134*A* and *B*, the I. Vs. for the various dentine areas in the moist slabs are placed on the left side of the vertical line indicating the approximate area read. The dentine slab outlines are sketched from the roentgenograms of the dry sections.

In Table CXVI are given the average D. Rs. in the various steps of penetrometers I and II in 2 films with dry tooth slabs taken at 30 kv., 30 ma., 15 inches, 0.85 sec., no filter, black-paper-wrapped films. The D-T curves (not shown) are

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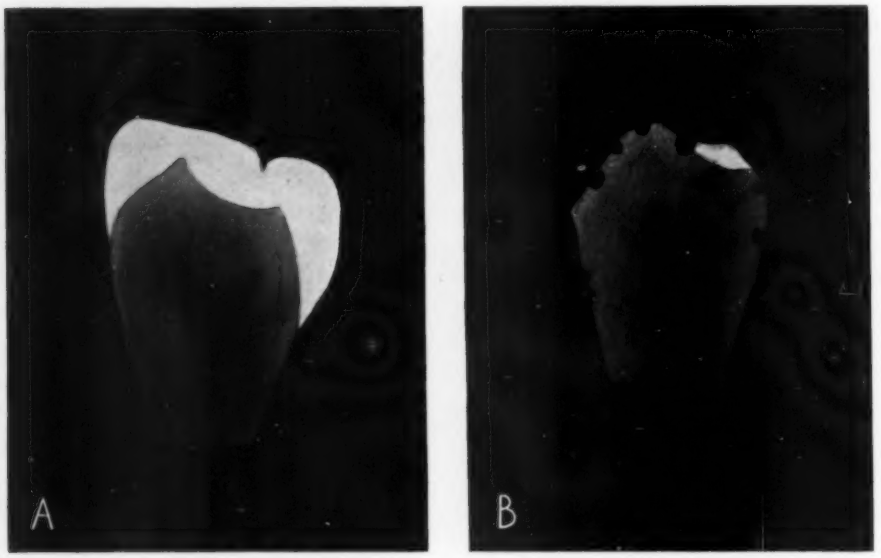

Fig. 133. Half-tone reproductions of the roentgenograms of tooth slabs No. 133 (compare Fig. 134*A*). *A* shows the initial appearance of the usual slab; *B* shows the appearance after the enamel has been removed and the dentine dried at 98° C. for three days.

typical, as are also the I. Vs. for the 4 teeth. The I. Vs. in the various areas of the dry dentine are placed on the right side of the vertical lines (see Figs. 134*A* and *B*). In tooth No. 27 (Fig. 134*A*) the coronal dentine is a somewhat better absorber of the roentgen radiation than the root dentine, but on the whole the I. Vs. from the

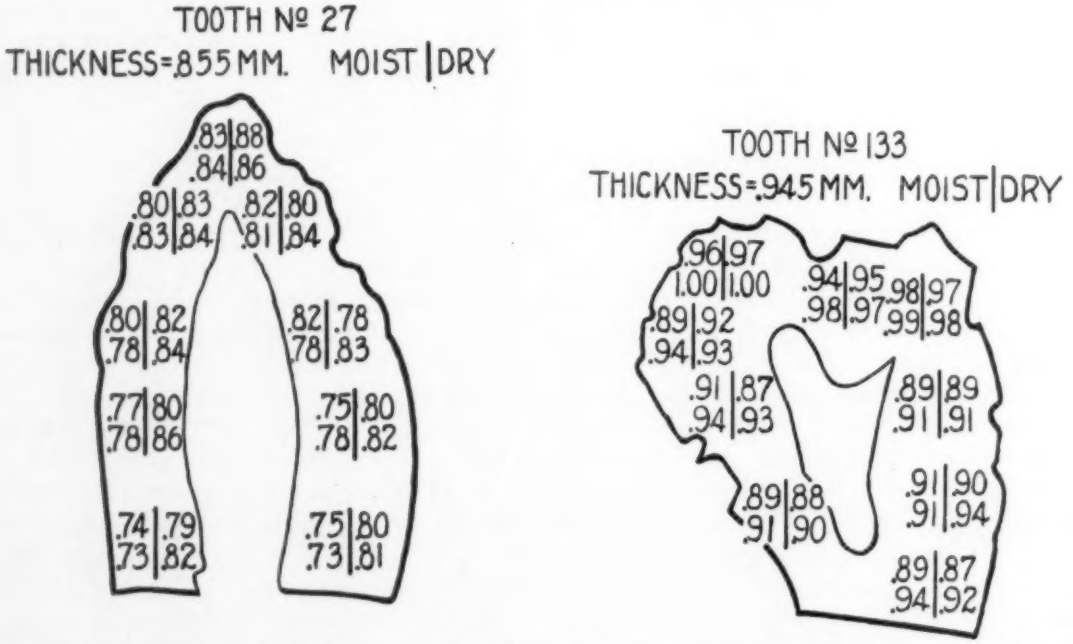

Fig. 134*A*. Outline drawings of the dentine slabs. The I. Vs. obtained in the moist state are placed on the left hand of the vertical lines; those obtained in the dried states on the right hand. The lines are placed approximately in the areas measured. Note that there is no consistent difference between I. Vs. on the moist or the dried slabs.

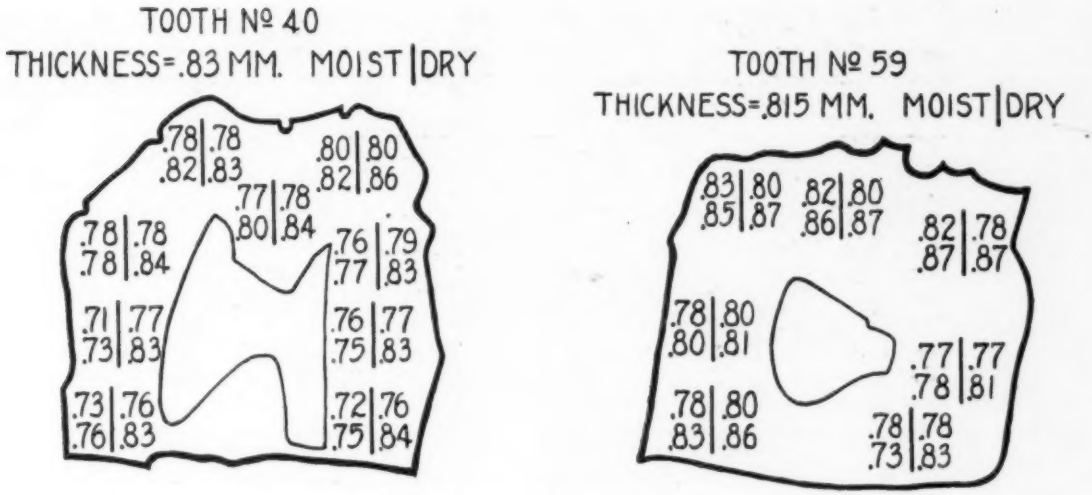

Fig. 134*B*

moist or dry slab are identical within the method error. In tooth No. 40 (Fig. 134*B*), the coronal dentine has a higher I. V. than the root dentine and with exception of the high data from film S34E88 (see Table cxvi), the I. Vs. are concordant. In tooth No. 59 (Fig. 134*B*), the data from film S34E88 are also high, the other I. Vs. being

Table CXV

MOIST TOOTH SLABS, 30 KV., 30 MA., 15 INCHES, 0.85 SEC., NO FILTER, BLACK-PAPER-WRAPPED FILM

Film S34E81			Film S34E82	
Step	D.R. Penetrometer I	D.R. Penetrometer II	D.R. Penetrometer I	D.R. Penetrometer II
1—	2.23	2.24	2.30	2.30
2—	1.80	1.78	1.81	1.80
3—	1.49	1.51	1.53	1.50
4—	1.26	1.28	1.33	1.26
5—	1.14	1.13	1.14	1.10
6—	1.00	0.98	1.00	0.98
7—	0.87	0.87	0.91	0.87
8—	0.81	0.82	0.83	0.81

Tooth No.	Dentine Area									Dentine Area								
	1	2	3	4	5	6	7	8	9	1	2	3	4	5	6	7	8	9
27	1.42	1.44	1.44	1.48	1.51	1.43	1.43	1.50	1.50	1.44	1.45	1.50	1.50	1.55	1.47	1.50	1.50	1.55
40	1.47	1.48	1.44	1.47	1.54	1.52	1.49	1.49	1.53	1.46	1.48	1.46	1.50	1.55	1.52	1.51	1.53	1.53
59	1.42	1.43	1.43	1.47	1.47	1.48	1.47			1.43	1.42	1.41	1.48	1.46	1.50	1.54		
133	1.30	1.32	1.29	1.36	1.35	1.36	1.36	1.35	1.36	1.30	1.32	1.31	1.35	1.35	1.38	1.38	1.38	1.35
Interpolated Values (mm. Al)																		
27	0.83	0.80	0.80	0.77	0.74	0.82	0.82	0.75	0.75	0.84	0.83	0.78	0.78	0.73	0.81	0.78	0.78	0.73
40	0.78	0.77	0.80	0.78	0.71	0.73	0.76	0.76	0.72	0.82	0.80	0.82	0.78	0.73	0.76	0.77	0.75	0.75
59	0.83	0.82	0.82	0.78	0.78	0.77	0.78			0.85	0.86	0.87	0.80	0.83	0.78	0.73		
133	0.96	0.94	0.98	0.89	0.91	0.89	0.89	0.91	0.89	1.00	0.98	0.99	0.94	0.94	0.91	0.91	0.91	0.94

TABLE CXVI

DRY TOOTH SLABS, 30 KV., 30 MA., 15 INCHES, 0.85 SEC. NO FILTER, BACK-PAPER-WRAPPED FILMS

Film S34E87			S34E88	
Step	D.R. I	Penetrometer II	Penetrometer I	II
1—	2.03	2.00	2.20	2.21
2—	1.63	1.63	1.78	1.80
3—	1.35	1.32	1.51	1.54
4—	1.14	1.16	1.28	1.32
5—	0.98	0.98	1.11	1.13
6—	0.85	0.86	0.99	1.01
7—	0.73	0.74	0.87	0.88
8—	0.68	0.69	0.80	0.81

Tooth No.	Dentine Area									Dentine Area								
	1	2	3	4	5	6	7	8	9	1	2	3	4	5	6	7	8	9
27	1.23	1.27	1.28	1.30	1.31	1.30	1.32	1.30	1.30	1.43	1.44	1.44	1.43	1.46	1.44	1.45	1.46	1.47
40	1.32	1.32	1.30	1.32	1.33	1.34	1.31	1.33	1.34	1.45	1.44	1.43	1.44	1.45	1.45	1.45	1.45	1.44
59	1.30	1.30	1.32	1.30	1.30	1.33	1.32		1.34	1.42	1.42	1.42	1.47	1.43	1.47	1.45		
133	1.16	1.18	1.16	1.20	1.24	1.23	1.22	1.21	1.24	1.30	1.33	1.32	1.36	1.36	1.38	1.39	1.35	1.37
Interpolated Values (mm. Al)																		
27	0.88	0.83	0.82	0.80	0.79	0.80	0.78	0.80	0.80	0.86	0.84	0.84	0.86	0.82	0.84	0.83	0.82	0.81
40	0.78	0.78	0.80	0.78	0.77	0.76	0.79	0.77	0.86	0.83	0.84	0.86	0.84	0.83	0.83	0.83	0.83	0.84
59	0.80	0.80	0.78	0.80	0.80	0.77	0.78			0.87	0.87	0.87	0.81	0.86	0.81	0.83		
133	0.97	0.95	0.97	0.92	0.87	0.88	0.89	0.90	0.87	1.00	0.97	0.98	0.93	0.93	0.90	0.91	0.94	0.92

concordant. In tooth No. 133 (Fig. 134*A*) there can be no significant difference shown in the I. Vs. moist or dry in the various areas measured.

TABLE CXVII

DENTINE SLABS

No.	Moist Wt.	Dry Wt.	Moisture Per cent
27	0.1007	0.0903	10.3
40	0.0941	0.0826	12.2
59	0.0702	0.0633	9.84
33	0.1100	0.0999	9.18

The moist weight of each slab (dentine with the enamel mostly chipped off), the dry weight and the percentage moisture are given in Table CXVII (data of M. L. LeFevre). The moisture percentages for tooth slabs No. 27, 40, 59, and 133 are 10.3, 12.2, 9.84, and 9.18 per cent, respectively; values which represent typical moisture contents for dentine.

In Table CXVIII are given the average I. Vs. for the moist and for the dry slabs. In tooth No. 27, all of the areas in the moist slab average 0.79 mm. Al; in the dry slab, 0.83 mm. Al; a difference less than the method error. In tooth No. 40, in the moist slab, the average I. V. is 0.77 mm. Al; in the dry slab, 0.81 mm. Al; a difference less than the method error. In tooth No. 59, in the moist slab, the average I. V. is 0.79 mm. Al; in the dry slab, 0.81 mm. Al; a difference less than the method error. In tooth No. 133, the practically identical I. Vs. are obtained of 0.93 mm. Al for the moist, 0.92 mm. Al for the dry slabs. It is noteworthy that the higher average I. Vs. for the dry slabs in teeth No. 27, 40, and 59 may be attributed to high values from film S34E88, nevertheless, the differences are all less than the method error. Comparing the average I. V. per unit thickness in the same tooth, moist *vs.* dry, it is evident that in no case is there a variation greater than 5 per cent.

From the discussion of the theory of the step tablet (Paper IV), the information is obtained that "for soft roentgen rays a chart showing the mm. of Al equivalent of bone, enamel, dentine (I. Vs.)—will be almost a chart showing the relative amounts of calcium present." The calcium accounts for about 75 per cent of the absorption in dentine at 0.7Å. Further, from Table XXXVIII under *Dentine*, it may be seen that the calcium is about 23 per cent of the moist slab and that the oxygen is about 48 per cent. Therefore, it is not at all surprising that removing approximately 10 per cent of the oxygen, i.e., leaving 38 per cent, should have a negligible effect on the total absorption from the standpoint of a method whose error in measuring total absorption is 5 per cent. Thus, we conclude that superficial drying (which does not approach to any extent complete drying) in the process of making roentgenograms, is of no consequence in influencing the magnitude of the I. Vs. for dentine.

B. D-T CURVES FOR ALUMINUM, DENTINE AND ENAMEL

A typical D-T curve represents a relation between the density produced in the film under the various thicknesses of Al and the actual thickness of the steps. It is possible with the data available from the experiment reported in Section c of this paper to draw a D-T curve for enamel and for dentine as well. However, when the D. Rs. (Table CXXII) in the enamel and in the dentine images are plotted against the thickness, the resulting graph shows somewhat aberrant readings for enamel and for dentine. This difficulty results from the fact that the different thicknesses of tooth slab were given slightly different exposures as may be seen from the D. R. in the penetrometers (Table CXXII). Therefore, in order to obtain comparable numbers in which exposure differences would not appear and which would permit us to plot D. Rs. under Al, enamel and dentine against thickness, we resorted to the expedient of choosing the fourth step (1.00 mm. Al) as a standard and comparing on each film the other steps of Al as well as the D. R. in enamel and in the dentine. Thus in film A43 (Table CXXII), the D. Rs. for Steps 2, 3, 4, 5, and 6 are 1.97 ,1.53, 1.20, 0.91, and 0.71. Comparing 2, 3, 4, 5 and 6 to 4 (1.20) the values are +0.77, +0.33, 0.00, −0.29, and −0.49. The average D. R. in the enamel in this film is about 0.60 which is −0.60 compared to Step 4. The average D. R. in the dentine is about 1.10 which is −.10 compared to Step 4. By studying each film in this way the data shown in Table CXIX are found.

When the average differences are plotted against the appropriate thickness as the step tablets of Al, enamel and dentine give the D-T curves shown in Figure 135. The dentine D-T curve is closely parallel to and slightly above the D-T curve for Al while the D-T curve for enamel is placed farther from and below the Al D-T curve. The

TABLE CXVIII

Moist Slabs	Av. I.V.	Av. I.V.	Av. I.V.	Av. I.V. per unit thickness
Tooth No.	S34E81	S34E82	Moist	Thickness (mm.)
27	0.79	0.79	0.79	0.92
40	0.76	0.78	0.77	0.95
59	0.80	0.78	0.79	0.97
133	0.92	0.95	0.93	0.98
Dry Slabs				
	S34E87	S34E88	Dry	Thickness
27	0.81	0.84	0.83	0.97
40	0.78	0.84	0.81	0.98
59	0.79	0.84	0.81	0.99
133	0.91	0.94	0.92	0.97

TABLE CXIX

D. RS. COMPARED TO STEP 4 AS A STANDARD

Penetrometer		Tooth Slab		
Step Al	Av. Diff.	Thickness	Av. Diff. En.	Av. Diff. Dent.
2	+0.77	1.20	−0.58	−0.10
3	+0.33	1.00	−0.51	+0.15
4	0.00	0.80	−0.34	+0.36
5	−0.27	0.60	−0.02	+0.70

places of the D-T curves for enamel and dentine relative to the Al D-T curve are predictable since dentine closely approximates the absorption properties of Al, while enamel absorbs a much larger amount of an incident beam than the same thickness of Al (see Paper IV). The smaller radius of curvature of the D-T curve for enamel is explained by the differences in the ratios of the absorption coefficients of enamel and Al at different thicknesses.

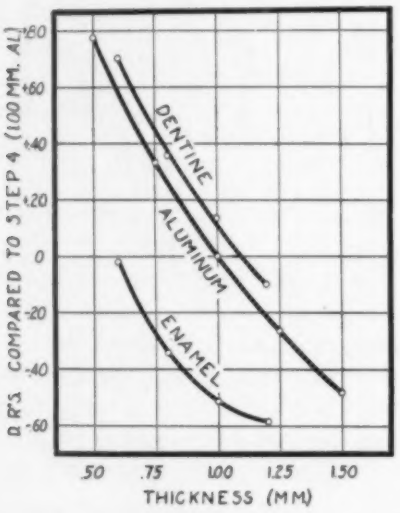

FIG. 135. D-T curves for dentine, aluminum and enamel. In effect, the data represent comparative step tablets of the two tissues. Note the close correspondence of dentine and aluminum, and that enamel gives a D-T curve with a smaller radius of curvature.

C. THE TOLERANCE IN THICKNESS OF THE TOOTH SLABS

The first attempt to estimate the tolerance within which the tooth slabs might vary in thickness without influencing the precision of the method was made by simultaneously exposing 6 tooth slabs whose thicknesses varied from 0.66 mm. ± 0.01 mm. to 0.82 mm. ±0.01 mm. In Table CXX are given the data obtained including the average D. Rs. on the various steps of the penetrometers, the D. Rs. in the enamel and in the dentine of the 6 teeth as well as the I. Vs. (in mm. Al) for the enamel and for the dentine of the 6 teeth. In Figure 136, the I. Vs. for the enamel and for the dentine are plotted as ordinates against the respective thickness for the 6 tooth slabs. The irregularity of the lines is due partly to the large method error, partly to the

TABLE CXX

THICKNESS OF TOOTH SLAB (FIRST ATTEMPT TO ESTIMATE TOLERANCE) 30 KV., 30 MA., 30 INCHES, 10 SEC. NO FILTER, BLACK-PAPER-WRAPPED FILM

Film 981		Tooth No.	Thickness	Densitometer Readings		I. V. (mm. Al)	
Step	D.R.			Enamel	Dentine	Enamel	Dentine
1—	1.88	106	0.66	1.40	1.61	1.22	0.84
2—	1.70						0.75
3—	1.56						0.72
4—	1.48						
5—	1.35	101	0.69	1.39	1.62	1.24	0.78
6—	1.30			1.38	1.62	1.26	0.78
7—	1.20						
8—	1.14	65	0.75	1.31	1.62	1.43	0.78
					1.62		0.78
					1.62		0.78
		98	0.80	1.32	1.62	1.41	0.78
					1.56		0.89
					1.60		0.82
		91	0.82	1.30	1.52	1.46	0.97
		77	0.82	1.30	1.56	1.46	0.89

fact that different teeth were used. When areas drawn (parallelograms, Fig. 136) whose altitude represents the difference in I. Vs. for enamel and for dentine which a difference of 0.02 in D. R. would make (see Paper v, G) it is seen that a difference of 0.034 mm. in the enamel thickness or of 0.047 mm. in the dentine thickness would be tolerated without affecting the precision of the method. (At the time the error in reproducibility was about 10 per cent.)

The second attempt to estimate the tolerance for the thickness of the tooth slabs was made on the same 6 tooth slabs as before but the method error having been reduced to about 6 per cent a better estimation was possible. The data on 2 films at 30 kv., 30 ma., 30 inches, 10 sec., no tube stand filter, and black-paper-wrapped films are given in Table cxxi. It may be noted that the greatest divergence between the I. Vs. using films 1234, and 1244 *for the enamel* is in the case of tooth No. 65 with a 4 per cent difference, *for the dentine* is in the case of tooth No. 91 with a 7 per cent difference. Still the lines connecting the I. Vs. for the enamel and for the dentine when they are plotted against the thickness of the respective tooth slab (not shown) are as irregular as those shown in Figure 136. By a procedure exactly similar to the former one, it is estimated that a difference of 0.02 mm. in the thickness of the enamel is sufficiently precise, and that a thickness difference of 0.035 mm. may be tolerated in the dentine.

The third attempt to estimate the tolerance for tooth slab thickness was radically different from the first two trials. The most easily ascribed reasons for the irregularity found in the I. V. thickness lines are the method error and the fact that different teeth are measured at the different thicknesses. The method error having been reduced to less than 5 per cent, we could only improve our conditions by measuring the same tooth at various thicknesses. For this purpose, 4 teeth of different sorts were selected all having intact enamel and no large carious areas. Slabs about 1.3 mm. thick were cut from their centers and ground planoparallel to 1.20 ±0.01 mm. by the Precision Optics Department of the Bausch and Lomb Optical Company. The exposures noted in Table cxxii were made and the slabs returned immediately to be

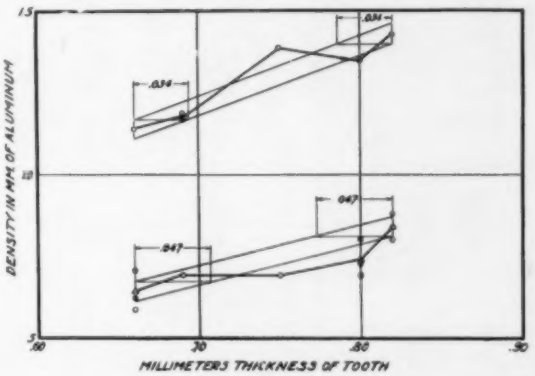

Fig. 136. The I. Vs. are plotted as ordinates against the thickness of enamel (upper broken curve) and of dentine (lower broken curve). The large apparent tolerance in thickness is due to the relative inaccuracy of the method (10 per cent) when this experiment was performed.

reground. In this way, exposures were made in the three days of March 27, 28, and 29, 1933, and at slab thicknesses of 1.20, 1.00, 0.80, and 0.60 mm., all within ±0.01 mm. of the stated thicknesses. Every effort was made to prevent drying or other changes of the tooth slabs. In Figure 137, the increasing density in the enamel and dentine images with decreasing thickness is illustrated.

In Table cxxii are given the data on the 4 tooth slabs exposed at 30 kv., 30 ma., 15 inches, 2 sec., no tube stand filter, black-paper-wrapped films. The table is subdivided into four parts in which the D. Rs. in the various steps of the penetrometers and in the enamel and the dentine are given along with the I. V. (mm. Al) for the 1.20, 1.00, 0.80, and 0.60 mm. thick tooth slabs. In each of the four subdivisions, the complete data for duplicate exposures are given. The eight D-T curves (not shown) are logarithmic, similar and in all respects typical for the exposure con-

TABLE CXXI

TOOTH SLAB THICKNESS (SECOND ATTEMPT TO ESTIMATE TOLERANCE) 30 KV., 30 MA., 15 INCHES, 2½ SEC., NO FILTER, BLACK-PAPER-WRAPPED FILMS

Film 1244		Tooth No.	Thickness (mm.)	Densitometer Readings		I. V. (mm. Al)	
				Enamel	Dentine	Enamel	Dentine
Step	D. R.						
1—	1.76	106	0.66	0.53	0.98	1.12	0.68
2—	1.20	101	0.69	0.50	0.96	1.17	0.96
3—	0.84	65	0.75	0.48	0.98	1.20	0.68
4—	0.59	98	0.80	0.43	0.89	1.30	0.75
5—	0.44	91	0.92	0.47	0.94	1.22	0.71
6—	0.36	77	0.82	0.44	0.89	1.28	0.75
7—	0.28						
8—	0.28						
Film 1243							
Step	D. R.						
1—	1.74	106	0.66	0.53	0.98	1.09	0.67
2—	1.17	101	0.69	0.47	0.98	1.18	0.67
3—	0.81	65	0.75	0.49	0.98	1.15	0.67
4—	0.57	98	0.80	0.42	0.83	1.28	0.77
5—	0.43	91	0.82	0.44	0.83	1.24	0.77
6—	0.34	77	0.82	0.44	0.80	1.24	0.80
7—	0.28						
8—	0.25						

ditions. The I. V. for the enamel average about 1.69 mm. Al for the thickness of 1.20 mm.; about 1.58 mm. Al for a thickness of 1.00 mm.; about 1.35 mm. Al for a thickness of 0.80 mm.; and about 1.04 mm. Al for a thickness of 0.60 mm. The I. V. for the dentine average about 1.09 mm. Al for a thickness of 1.20 mm.; about 0.92 mm. Al for a thickness of 1.00 mm.; about 0.77 mm. Al for a thickness of 0.80 mm.; and about 0.58 mm. Al for a thickness of 0.60 mm. By dividing the average I. Vs. for enamel and for dentine by the appropriate thicknesses a comparative ratio is obtained, the I. V. per unit thickness (Table CXXIII). These average I. Vs. are plotted as ordinates against the proper thicknesses in Figure 138. The average I. Vs. for enamel fall on a smooth curve which rises less rapidly with increasing thickness of tooth slab. The average I. Vs. for dentine lie on a straight line. From these lines it may be estimated that a tolerance of about ±0.02 mm. for the enamel, and of ±0.01 mm.

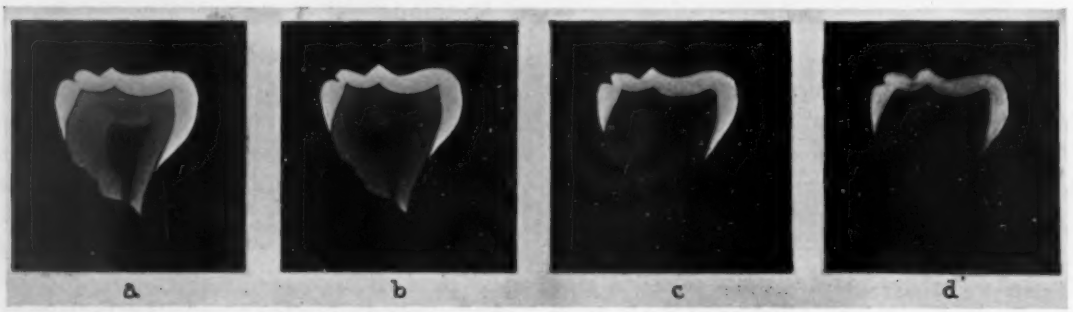

FIG. 137. Note the increase of density in the enamel and dentine images with decrease in thickness of the slab. The thicknesses are *a*, 1.20 mm.; *b*, 1.00 mm.; *c*, 0.80 mm., and *d*, 0.60 mm.

TABLE CXXII

THICKNESS OF TOOTH SLAB (THIRD ATTEMPT TO ESTIMATE TOLERANCE) 30 KV., 30 MA., 15 INCHES, 2 SEC. NO FILTER, BLACK-PAPER-WRAPPED FILM

	1.20 mm. thick				1.00 mm. thick				0.80 mm. thick				0.60 mm. thick			
	Film A_{43}		Film B_{41}		Film A_{44}		Film B_{42}		Film A_{45}		Film B_{43}		Film A_{46}		Film B_{44}	
Step	D. R.	I. V.	D. R.	I. V.	D. R.	I. V.	D. R.	I. V.	D. R.	I. V.	D. R.	I. V.	D. R.	I. V.	D. R.	I. V.
1 —	2.65		2.60		2.8		2.77		2.74		2.75		2.8		2.71	
2—	1.97		1.97		2.29		2.09		2.05		2.05		2.19		2.04	
3—	1.53		1.53		1.84		1.64		1.62		1.62		1.73		1.61	
4—	1.20		1.22		1.49		1.32		1.29		1.28		1.40		1.27	
5—	0.91		0.96		1.26		1.02		1.01		1.03		1.12		1.02	
6—	0.71		0.74		1.04		0.84		0.82		0.79		0.89		0.78	
7—	0.53		0.58		0.83		0.64		0.61		0.61		0.71		0.62	
8—	0.47		0.50		0.73		0.57		0.53		0.56		0.66		0.55	
Tooth A.																
En.	0.65	1.59	0.65	1.68	0.97	1.56	0.81	1.55	0.94	1.33	0.97	1.32	1.36	1.06	1.27	1.03
Dent.	1.13	1.07	1.12	1.10	1.61	0.96	1.38	0.97	1.65	0.77	1.57	0.82	2.07	0.61	1.95	0.60
B.																
En.	0.65	1.59	0.61	1.78	0.97	1.56	0.83	1.53	0.97	1.30	0.93	1.36	1.36	1.06	1.26	1.03
Dent.	1.04	1.14	1.09	1.13	1.68	0.91	1.49	0.89	1.70	0.74	1.57	0.82	2.13	0.57	0.98	0.58
C.																
En.	0.64	1.61	0.60	1.80	0.97	1.56	0.84	1.50	0.93	1.34	0.93	1.36	1.36	1.06	1.25	1.04
Dent.	1.11	1.08	1.14	1.08	1.72	0.88	1.52	0.87	1.67	0.76	1.63	0.78	2.13	0.57	1.95	0.60
D.																
En.	0.60	1.70	0.61	1.78	0.95	1.56	0.81	1.55	0.98	1.29	0.93	1.36	1.39	1.03	1.27	1.03
Dent.	1.13	1.07	1.16	1.07	1.63	0.94	1.46	0.92	1.70	0.74	1.69	0.75	2.17	0.55	1.97	0.58

for the dentine is possible without exceeding the variation produced by a difference of 0.02 density units in any D. R.

D. REPRODUCIBILITY OF THE METHOD

In order to provide an adequate test of the reproducibility of the method, some object should be chosen which would have

TABLE CXXIII

AVERAGE I. V. *vs.* THICKNESS FOR ENAMEL AND DENTINE

Thickness	Enamel	Dentine
1.20	1.41	0.91
1.00	1.58	0.92
0.80	1.69	0.96
0.60	1.73	0.97

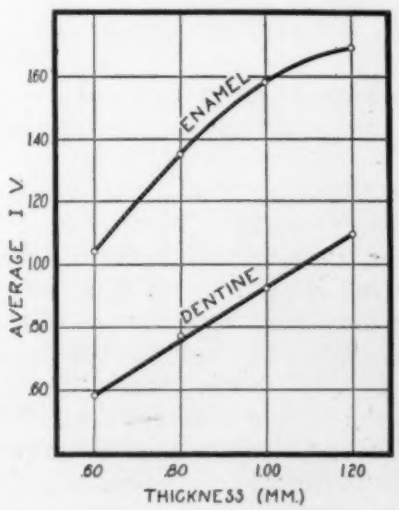

FIG. 138. The average I. Vs. for enamel lie on a smooth curve which rises less rapidly with increasing thickness of enamel. The average I. Vs. for dentine lie, as would be predicted, on a straight line.

a large enough area of homogeneous enamel-like material to remove the possibility of errors (*a*) from densitometer characteristics, (*b*) from the development effects noted in a previous paper (Paper VIII) which give brighter lines along the peripheral portion of the enamel image, or (*c*) from actual variations in enamel radiopacity.

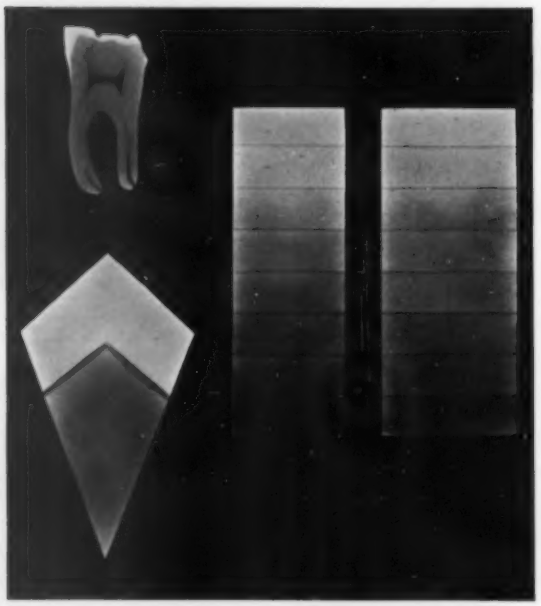

FIG. 139. The "phantom" tooth is composed of Zn-alloy for the enamel part and pure Al for the dentine part. The arrangement of the tooth slab, "phantom," Al penetrometer and alloy penetrometer is exactly as on the films. The matching Al and alloy penetrometers were placed in similar fashion on the left of the slab and phantom.

The test object should also have a dentine-like material which would be homogeneous, thus removing the errors in reproducibility of I. Vs. in the dentine, due to the frequently found areas of changed radiopacity. The object selected for this test was a "phantom" tooth (Fig. 139) of which the enamel area was made from the alloy of 6 per cent zinc and 94 per cent aluminum described previously (Paper IV) and the dentine area of pure aluminum. The alloy should give an "alloy value," i.e., the thickness of the alloy step tablet giving the same density, of exactly its own thickness; the aluminum should give an "interpolated value," i.e., the thickness of the Al step tablet giving the same density, equal to its own thickness. In addition, a tooth was selected and roentgenographed together with the phantom and the penetrometers.

In order that the test be made as rigorous as possible, a trained roentgen-ray technician but one who had not had previous acquaintance with the method was selected to take the roentgenograms and develop them.* Further a trained roentgenographer accustomed to densitometric work but without previous acquaintance with this method was selected to obtain the densitometer readings and interpolate the data. The arrangement for roentgenographing consisted of the two alloy or "enamel" penetrometers placed beside the two aluminum or "dentine" penetrometers which in turn enclosed the tooth section and the "phantom" tooth. Figure 139 shows the tooth slab, the "phantom," one aluminum and one alloy penetrometer; the other two penetrometers were on the left of the slab and "phantom" and are not shown. The comparative sizes of slab, phantom, and penetrometers may be estimated from the figure.

A series of 100 exposures were made in 4 batches on four days. Two exposures were made on each 5″×7″, black-paper-wrapped "dupli-tized x-ray film." All the exposures were made at 30 kv., 30 ma., 30 inches, 16/25 seconds with a round focus tube. Using a Victoreen r-meter, model 70, this combination equals an exposure of 33.8 r with readings averaging 52.8 r per second at the film level. Sample data from one day included the following:

The developer was prepared the day before. The films were developed at 18.3° C. for 5 minutes with hand agitation, fixed for 5 minutes in fresh fixer and washed for 15 minutes in running water. Room

* It is a pleasure to acknowledge the valuable services of Mr. Herbert Mermagen of the Division of Radiology of the Department of Medicine of the Strong Memorial Hospital, and of Mr. H. F. Sherwood of the Eastman Kodak Company.

temperature, 74° F.; room humidity, 47 per cent.

Along with each batch of films, sensitometric strips were developed. The densitometer readings on each strip were plotted in the typical H and D curves and proved by the normal gamma value obtained that the development procedure was correct photographically.

Three densitometer readings were obtained in each step of each penetrometer. The D. Rs. for a given step were averaged for the two alloy penetrometers and separately for the two aluminum penetrometers. At least three D. Rs. each were made in the "phantom" enamel (alloy) and dentine (aluminum) and in the enamel and the dentine of the tooth slab. The D-T curves were plotted and the following interpolations performed:

(1) I. Vs. for aluminum and alloy of the "phantom"; for dentine and enamel of the tooth slab; i.e., from the aluminum penetrometer D-T curves.

(2) Alloy values for the alloy of the "phantom" and for the enamel of the tooth slab; i.e., from the alloy penetrometer D-T curves.

Since the entire test included over 10,000 D. Rs. and about 2,000 I. Vs. only a summary of the data is given in Table CXXIV. In the first column is given the film number, the A and B referring to the two exposures on each 5″×7″ film. In the second and third columns, the gradients, i.e., the density differences from the left hand side of the left penetrometer to the right hand side of the right penetrometer, are given, showing the constancy of the films from the left to right. In the fourth and fifth columns, the density ranges of the penetrometers, i.e., the differences in density from Step 1 to Step 8, are given, showing the constancy of the films up and down. In the sixth to ninth columns, the average D. Rs. in the enamel, dentine, alloy, and aluminum (the latter of the "phantom") are given. In the tenth to thirteenth columns, the average I. Vs. for enamel, dentine, alloy, and aluminum (the latter two of the "phantom") are given. In the fourteenth and fifteenth columns, the average alloy value of the enamel (tooth slab) and of the alloy of the "phantom" are given.

The general averages from Table CXXIV are given in Table CXXV. The average interpolated values for enamel range from 0.86 to 1.18 mm. Al. The general average for the enamel I. Vs. is 1.02 mm. Al, and since the slab thickness was 0.59 ± 0.01 mm., on conversion, therefore, the average I. V. per unit thickness is 1.72 mm. Al.

The average interpolated values for dentine range from 0.45 to 0.74 mm. Al. The general average for the dentine I. Vs. is 0.58 mm. Al, or (see above) the average I. V. per unit thickness is 0.98 mm. Al.

The average interpolated values for the alloy (enamel) of the "phantom" range from 1.44 to 1.85 mm. Al. The general average for the alloy I. Vs. is 1.63 mm. Al and since the alloy was 1.01 mm. thick, the average I. V. per unit thickness is 1.63 mm. Al.

The average interpolated values for the aluminum (dentine) of the "phantom" range from 0.87 to 1.16 mm. Al. The general average for the aluminum I. Vs. (discarding all values differing from the average by 3.5 times the standard deviation) is 0.99 mm. Al., and since the aluminum was 1.00 mm. thick, the average I. V. per unit thickness is 0.99 mm. Al.

The average alloy values for the enamel of the tooth slab range from 0.50 to 0.74 mm. alloy. The general average for the enamel alloy values is 0.62 mm. alloy and (see above) the average alloy value per unit thickness is 1.05 mm. alloy.

The average alloy values for the alloy (enamel) of the "phantom" range from 0.85 to 1.11 mm. alloy. The general average of the alloy values is 0.97 and (see above) the average alloy value per unit thickness is 0.96 mm. alloy.

The ranges of the standard deviations about the averages are about 7 units for the enamel I. Vs., and about 6 units for the dentine I. Vs. Since the test of the method must be found in the reproduci-

TABLE CXXIV

Film No.	Grad. En.	L-R. Dent.	Range En.	O.R. 1–8 Dent.	Average D. R.			Average I. V.				Alloy V.		
					En.	Dent.	Alloy.	Al.	En.	Dent.	Alloy.	Al.	En.	Alloy.
24A	.01	.06	.96	.92	1.11	1.39	.83	1.09	.94	.54	1.56	.96	.58	.96
24B	.01	.02	.97	.97	1.06	1.35	.84	1.17	1.06	.61	1.55	.89	.63	.96
25A	.02	.06	.98	.91	1.12	1.40	.84	1.11	.93	.50	1.55	.95	.60	.97
25B	.02	.04	.99	.95	1.08	1.34	.81	1.11	1.02	.59	1.71	.95	.61	.96
26A	.01	.03	.99	.94	1.17	1.40	.85	1.12	.91	.57	1.63	1.00	.51	.94
26B	.02	.01	1.07	.97	1.15	1.39	.87	1.14	1.00	.66	1.59	1.03	.60	.97
27A	03	.04	1.05	.96	1.18	1.42	.87	1.14	.95	.59	1.57	1.02	—	.98
27B	.04	.06	1.10	.99	1.13	1.36	.86	1.15	1.02	.66	1.60	.98	.62	.97
28A	.03	.03	1.03	.95	1.16	1.40	.83	1.12	.90	.54	1.63	.98	.54	.98
28B	.02	.03	1.02	.97	1.08	1.34	.82	1.11	1.05	.61	1.70	.99	.63	1.00
29A	.01	.03	1.00	.92	1.11	1.34	.82	1.11	.99	.61	1.75	.97	.57	.98
29B	.02	.05	1.02	.92	1.12	1.34	.83	1.12	.96	.61	1.63	.96	.61	1.00
30A	.08	.00	1.01	.98	1.15	1.44	.85	1.12	.97	.54	1.68	1.03	.58	.97
30B	.03	.00	1.01	.94	1.10	1.39	.84	1.10	1.01	.56	1.59	1.00	.60	.98
31A	.03	.01	1.02	.98	1.18	1.45	.87	1.15	.97	.56	1.71	1.02	—	1.00
32B	.03	.01	.99	.97	1.17	1.41	.87	1.17	.95	.58	1.55	.96	.55	.92
32A	.02	.04	.99	.93	1.14	1.39	.83	1.09	.94	.55	1.66	1.05	.56	1.05
32B	.03	.02	1.03	.97	1.14	1.43	.88	1.14	.94	.61	1.59	1.05	.62	.92
33A	.03	.01	1.00	.95	1.20	1.51	.89	1.13	.91	.47	1.60	1.07	.58	.99
33B	.02	.02	1.02	1.00	1.13	1.38	.89	1.14	.98	.62	1.47	.95	.57	.90
34A	.02	.02	1.01	.95	1.24	1.52	.90	1.16	.89	.48	1.66	1.03	.54	.98
34B	.03	.01	1.01	.97	1.14	1.39	.87	1.18	1.01	.62	1.64	.94	.60	1.00
35A	.07	.01	1.06	.93	1.20	1.38	.90	1.17	.97	.55	1.70	1.02	.62	1.00
35B	.03	.01	1.07	1.03	1.12	1.43	.85	1.15	.99	.56	1.52	.94	.63	.99
36A	.02	.02	.95	.90	1.13	1.39	.82	1.10	.93	.51	1.68	.99	.55	.98
36B	.06	.06	.98	.85	1.01	1.29	.80	1.04	1.10	.62	1.62	1.03	.67	.99
37A	.04	.01	1.02	.97	—	1.46	.81	1.10	—	.49	1.77	1.00	—	.98
38A	.06	.03	1.08	1.03	1.10	1.43	.83	1.13	1.07	.59	1.67	1.01	.64	.99
38B	.02	.04	1.11	1.07	1.14	1.42	.83	1.17	1.04	.65	1.68	1.00	.63	1.01
39A	.02	.01	1.08	1.00	1.13	1.43	.80	1.10	.86	.53	1.69	.99	.58	1.00
39B	.03	.02	1.12	1.05	1.08	1.36	.83	1.13	1.07	.64	1.65	.97	.63	.96
40A	.07	.04	1.05	.95	1.11	1.39	.84	1.10	.99	.56	1.66	1.01	.60	.98
40B	.03	.00	1.08	1.04	1.10	1.37	.82	1.13	1.06	.65	1.67	1.02	.63	.85
41A	.01	.01	.94	.87	1.17	1.44	.84	1.12	.89	.48	1.74	.98	.50	.97
41B	.01	.01	1.03	.99	1.07	1.34	.84	1.14	1.09	.63	1.63	.94	.65	.97
42A	.01	.02	1.06	.97	1.14	1.46	.82	1.11	.95	.49	1.65	1.01	.58	1.00
42B	.01	.01	1.10	1.05	1.01	1.28	.81	1.12	1.18	.72	1.47	.98	.70	.98
43A	.11	.01	.93	.85	1.09	1.36	.85	1.10	1.02	.57	1.67	1.00	.59	.98
43B	.03	.05	.97	.93	1.04	1.35	.83	1.09	1.09	.57	1.74	1.02	.65	.97
44A	.13	.06	1.00	.92	1.20	1.49	.90	1.20	.96	.54	1.74	.96	.55	1.00
44B	.01	.02	1.01	.97	1.09	1.39	.92	1.21	1.18	.67	1.57	.96	.67	.92
45A	.02	.01	.99	.88	1.19	1.47	.92	1.15	1.03	.54	1.80	1.12	.63	1.01
45B	.04	.01	1.01	.95	1.21	1.51	.92	1.18	.93	.48	1.55	.98	.58	.95
46A	.05	.06	1.02	.94	1.20	1.55	.99	1.25	1.17	.58	1.78	1.06	.72	1.04
46B	.04	.11	1.05	.94	1.18	1.45	1.00	1.27	1.14	.66	1.56	.96	.72	.96
47A	.05	.02	1.00	.92	1.27	1.56	.97	1.24	.94	.50	1.69	1.00	.56	.97
47B	.04	.03	1.04	.97	1.16	1.38	.94	1.22	1.17	.72	1.59	.97	.65	.96
48A	.05	.03	.97	.91	1.16	1.47	.94	1.18	1.07	.55	1.66	1.04	.67	1.00
48B	.07	.06	1.00	.98	1.10	1.38	.90	1.19	1.12	.64	1.56	.94	.68	.98
49A	.01	.00	1.02	.92	1.28	1.59	1.03	1.26	1.00	.53	1.62	1.03	.60	.89
49B	.01	.01	1.02	.93	1.24	1.58	1.03	1.26	1.07	.53	1.62	1.04	.62	.90
50A	.06	.04	1.02	.93	1.29	1.58	1.00	1.27	1.00	.55	1.72	1.03	.58	.96
50B	.02	.06	1.03	1.00	1.21	1.49	1.00	1.28	1.08	.63	1.56	1.00	.67	.96
51A	.09	.00	.97	.86	1.18	1.48	.94	1.18	.99	.51	1.68	.97	.60	.92
51B	.01	.03	.97	.98	1.09	1.38	.94	1.20	1.13	.64	1.50	.91	.67	.89
52A	.03	.03	.97	.97	1.14	1.39	.88	1.15	.96	.58	1.56	.94	.57	.97
52B	.02	.03	.90	.83	1.16	1.46	.94	1.18	.98	.46	1.54	.93	.59	.94
1A	.05	.00	1.03	.95	1.12	1.35	.81	1.09	.89	.56	1.58	.94	.53	.95
1B	.08	.05	1.04	.99	1.12	1.39	.81	1.11	.94	.55	1.64	.95	.56	.97
2A	.08	.12	1.08	1.01	1.14	1.45	.84	1.13	.94	.51	1.59	.96	.57	.94
2B	.00	.05	1.10	1.08	1.18	1.51	.88	1.17	1.00	.57	1.62	1.05	.58	.98

Table CXXIV—*Continued*

Film No.	Grad. En.	L-R. Dent.	Range En.	O.R. 1–8 Dent.	Average D. R.			Average I. V.				Alloy V.		
					En.	Dent.	Alloy.	Al.	En.	Dent.	Alloy.	Al.	En.	Alloy.
3A	.05	.00	1.17	1.04	1.12	1.44	.80	1.12	.97	.55	1.71	.97	.60	1.04
3B	.08	.01	1.20	1.13	1.12	1.50	.87	1.21	1.07	.57	1.61	.94	.65	1.00
4A	.04	.04	1.14	1.04	1.20	1.50	.95	1.22	1.04	.63	1.49	1.00	.63	.94
4B	.05	.01	1.10	.99	1.25	1.55	.96	1.22	1.03	.58	1.61	1.07	.61	.96
5A	.02	.00	1.16	1.07	1.29	1.59	1.04	1.33	1.05	.63	1.56	.99	.64	.94
5B	.05	.04	1.15	1.10	1.37	1.67	1.06	1.37	1.04	.61	1.67	1.03	.61	.96
6A	.02	.06	1.08	1.02	—	1.55	.90	1.21	—	.53	1.70	.99	—	.98
6B	.12	.00	1.11	1.06	1.22	1.48	.96	1.24	1.02	.65	1.51	.99	.60	.92
7A	.05	.02	1.11	1.08	1.25	1.01	1.02	1.30	1.06	.56	1.53	.98	.63	.90
7B	.13	.03	1.08	1.02	1.20	1.49	.88	1.19	.98	.58	1.66	.99	.66	1.09
8A	.01	.03	1.10	1.04	1.12	1.44	.87	1.20	1.13	.63	1.70	.99	.67	1.01
8B	.03	.03	1.08	1.00	1.19	1.47	.90	1.20	.94	.55	1.61	.92	.57	.92
9A	.04	.02	1.07	1.01	1.07	1.33	.80	1.07	.99	.60	1.61	.98	.58	.95
9B	.04	.02	1.14	1.06	1.12	1.47	.81	1.13	1.01	.55	1.68	.99	.61	1.03
10A	.02	.03	1.05	.98	1.08	1.39	.81	1.11	1.03	.56	1.72	.99	.61	.98
10B	.05	.07	1.11	1.05	1.10	1.38	.83	1.12	1.05	.64	1.66	1.02	.62	1.01
11A	.01	.05	1.07	1.00	1.10	1.39	.82	1.14	1.07	.63	1.77	1.00	.67	1.07
11B	.03	.01	1.11	1.00	1.16	1.47	.86	1.17	.99	.55	1.64	.98	.62	1.02
12A	.06	.03	1.15	1.10	1.28	1.59	.97	1.30	1.01	.56	1.65	.98	.57	.92
12B	.05	.05	1.19	1.05	1.30	1.57	.98	1.27	.96	.60	1.61	1.01	.61	.98
13A	.10	.06	.96	.88	1.30	1.56	1.01	1.27	.93	.53	1.73	.97	.55	.98
13B	.04	.04	.98	.93	1.20	1.48	.98	1.26	1.15	.64	1.70	1.00	.68	1.01
14A	.02	.02	.95	.85	1.20	1.51	.91	1.15	.98	.47	1.80	1.10	.60	1.05
14B	.01	.00	.99	.98	1.13	1.39	.94	1.25	1.16	.70	1.64	.92	.73	1.01
15A	.09	.07	.96	.90	1.20	1.46	1.01	1.25	1.15	.66	1.68	1.04	.64	.97
15B	.09	.07	.96	.90	1.20	1.46	1.01	1.25	1.15	.66	1.68	1.04	.64	.97
16A	.06	.07	.97	.93	1.20	1.49	1.00	1.26	1.04	.55	1.53	.91	.60	.86
16B	—	.04	1.01	.96	1.16	1.44	.94	1.23	1.18	.69	1.74	1.03	.69	1.03
17A	.03	.04	.96	.90	1.17	1.45	.98	1.27	1.09	.63	1.61	.90	.62	.90
17B	.06	.06	.94	.92	1.12	1.44	.94	1.18	1.11	.57	1.55	.99	.64	.96
18A	.02	.02	.97	.88	1.24	1.54	.97	1.20	.96	.50	1.70	1.05	.63	1.05
18B	.04	.02	.99	.96	1.16	1.45	.98	1.25	1.10	.64	1.59	.95	.69	1.00
19A	.07	.06	.99	.94	1.20	1.49	.97	1.26	1.05	.61	1.66	.94	.63	.99
19B	.03	.01	.96	.94	1.14	1.46	.93	1.17	1.08	.55	1.61	1.04	.65	.95
20A	.06	.08	.95	.97	1.30	1.63	1.05	1.29	1.01	.51	1.60	1.02	.61	.94
20B	.03	.03	.96	.99	1.22	1.55	1.02	1.28	1.13	.59	1.61	1.01	.66	.94
21A	.01	.01	.98	.92	1.22	1.49	1.00	1.23	1.00	.59	1.57	.99	.64	.96
21B	.02	.01	.97	.97	1.19	1.52	1.02	1.25	1.15	.59	1.57	1.01	.67	.91
22A	.01	.02	.99	.86	1.29	1.58	1.10	1.32	1.03	.52	1.58	.96	.63	.90
22B	.03	.05	1.00	.92	1.23	1.51	1.05	1.29	1.10	.62	1.62	.97	.68	.96
23B	.05	.03	.85	.77	1.18	1.38	.93	1.19	.98	.59	1.77	.96	.58	.97

bility of the values for the aluminum of the "phantom" in terms of the aluminum penetrometers, it is interesting to note that the standard deviation for the aluminum I. Vs. is 4.4 and for the alloy values is 3.5. Of the 306 I. Vs. averaged for the alumi-

Table CXXV

General Average from Table CXXIV

Penetrometer					Average I. V.				Average Alloy V.	
Gradient Left-right			Range Steps 1–8		Enamel	Dentine	Alloy	Al	Enamel	
No. of values	Enamel 102	Dentine 102	Enamel 102	Dentine 102	100	102	101	102	98	102
Total	3.94	3.12	105.77	98.48	102.22	59.08	165.10	97.95	60.38	99.02
Average	0.038	0.031	1.037	0.965	1.022	0.579	1.634	0.960	0.616	0.971

num, 213 fall between 0.94 and 1.02; of the 302 alloy values averaged for the alloy of the "phantom," 209 fall between 0.93 and 1.01. These values show over-all ranges of the standard deviations of about 8 per cent of the averages and indicate the precision of the method under conditions which were believed to be a rigorous test.

Since the first test of the method should involve the comparison of the density of the image of a given thickness or aluminum alloy against the density thickness curves of penetrometers made of aluminum or alloy, respectively, it is especially interesting to note that the reproducibility of the method is better for this comparison (the "phantom" tooth) than for the actual tooth slab. Thus, the slightly wider variations found for I. Vs. of dentine and enamel (Table CXXIV) may be attributed to variations, histological, chemical, or physical, in the tissues or to the effect of other factors enumerated above.

CONCLUSIONS

1. Drying of the surface of the tooth slab during casual roentgenographic technique does not influence the I. Vs. Removal of all the moisture causes changes in the I. Vs. of less than the method error.

2. By grinding down the same tooth slab through a series of successive thicknesses and making serial roentgenograms of each thickness (1.20–0.60), it is possible with a densitometric method error of less than 5 per cent to estimate that a tolerance of ±0.02 mm. in thickness for enamel and ±0.01 mm. for dentine is possible without exceeding the variation in density of 0.02 units (the readable density reproducibility).

3. In a series of 100 identical roentgenograms of the penetrometers, a tooth slab and a "phantom" tooth, the reproducibility of the method in the hands of other than our own group (H. M. and H. F. S.) is shown to involve an error of about 5 per cent.

THE ADVANTAGES OF NON-SCREEN ROENTGENOGRAPHY*

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IN HIS original report on the discovery of x-rays in 1895, Roentgen stated that he considered it of great importance that the newly discovered rays could be recorded on photographic plates. Roentgen wrote in this now famous publication that every one of the more important observations he made with these x-rays had been controlled and verified by making photographic records.

Today we realize that the emphasis placed by the discoverer on this particular characteristic of roentgen radiation was very foresighted. Roentgenography has become a highly important branch of medicine and photographic roentgen-ray images are an outstanding part of modern medical and dental diagnosis.

The action of roentgen rays on photographic emulsions is at the same time similar to and different from the action of visible light on such materials. Like visible light the roentgen radiation forms a latent image in the photographic emulsion layer. This latent image can be developed and thereby converted into a permanent silver image. However, it was soon recognized that the exposure of photographic emulsions with the radiation from roentgen tubes was much less efficient than the exposure with visible light of much lower energy.

In exposing photographic film with roentgen rays, by far the greater part of the radiation penetrates the light sensitive emulsion layer without contributing to the formation of the latent image. Only a small portion of the roentgen-ray energy is absorbed by the emulsion and can therefore be utilized in producing a latent image. In other words, the photographic emulsion, while being highly sensitive to visible light, has only a comparatively low sensitivity for direct roentgen radiation.

It was discovered by Pupin, however, that this wasted roentgen-ray energy which penetrates the photographic layer without being utilized can be transferred into photographically useful radiation by the use of screens containing calcium tungstate crystals. The roentgen rays are absorbed by the calcium tungstate particles in the screen and emitted again in a transformed radiation of longer wave length as fluorescent light.

The intensity of the fluorescent light emanating from these tungstate particles is in direct proportion to the roentgen-ray energy with which the particles have been exposed. Using these screens in close contact with the film, which is highly sensitive to this fluorescent light, it is possible to obtain roentgen-ray images on the photographic film with much less roentgen-ray energy or with a much shorter exposure time. The calcium tungstate screens have become generally known and are today universally used under the name of "intensifying screens."

A roentgenogram made with the use of intensifying screens is thus a photographic image which has been obtained by two different forms of radiation: first, the primary roentgen rays having a wave length of from 0.3 to 0.6 Ångström units, and second, by the fluorescent light which has been generated by the impact of roentgen rays on the intensifying screen. This fluorescent or visible light has a wave length of about 4,000 to 5,000 Ångström units and contributes by far the greater part to the formation of the roentgen-ray image on the film. In most cases the direct roentgen radiation contributes only

* From the Agfa Ansco Corporation, Binghamton, N. Y.

to the blackening of the areas outside the roentgen-ray subject.

The invention of intensifying screens was the first important improvement in the technique of making photographic roentgen-ray images. With the introduction of roentgen films coated with emulsion on both sides, the use of screens became even more advantageous because intensifying screens could then be used on both sides of the film, transforming still more of the otherwise not utilized direct roentgen radiation into photographically more effective fluorescent light.

Constant improvements have also been made in the development of new and better high speed roentgen-ray emulsions as well as in the perfection of intensifying screens with greater intensifying effect and improved definition. The advancements in both these lines have made it possible to cut exposure energy and exposure time to a level, where repeated roentgenograms can be made on the same patient without any harmful effect.

It has, of course, been known all the time that with the use of intensifying screens a very noticeable sacrifice has to be made in regard to detail sharpness and definition. Realizing this distinct disadvantage, intensifying screens have rarely been used for smaller extremities, where the penetration is sufficient to allow the use of regular roentgen films without screens, thereby obtaining sharper roentgenograms with more detail and of greater diagnostic value.

If we consider briefly the reasons for the lack of detail sharpness, caused by the intensifying screens, it will become quite obvious that there is little hope that this deficiency of the intensifying screen technique can be entirely overcome.

There are three principal reasons why intensifying screens cause loss in detail rendition. Taking as an example calcium tungstate which is still one of the most effective substances used in intensifying screens, it is important to realize that the intensifying effect of screens containing this mineral depends upon the grain size of the individual tungstate crystals. The smaller grains give a lower intensifying factor, while larger grains show much stronger intensifying effects. Very small tungstate crystals are used for the so-called detail screen types, while larger crystals are used for the par speed and still larger ones for the high speed screens.

In Figure 1 photomicrographs of the tungstate grains are shown in comparison with the grain size of a high speed roentgen-ray emulsion. It can be seen that the detail screen (*B*) has a much finer grain compared to the high speed screen (*C*), but even the so-called detail screen still has very noticeably larger grains than the high speed roentgen-ray emulsion (*A*). The grain of the high speed screen is from about five to ten times larger than that of the photographic emulsion. Considering the functions of these individual tungstate grains during a roentgen-ray exposure, it can be easily seen why the grain size of these screens is of such great importance for the rendition of detail.

When exposed by roentgen radiation, the individual tungstate grains function as numerous little sources of fluorescent light. The larger grains, of course, will expose from about five to ten silver halide grains, covering the same area as the tungstate grain, while the original beam of radiation probably would have struck only one or two of these silver halide grains. As a result the details are spread over a somewhat larger area and are therefore less sharp in their outlines.

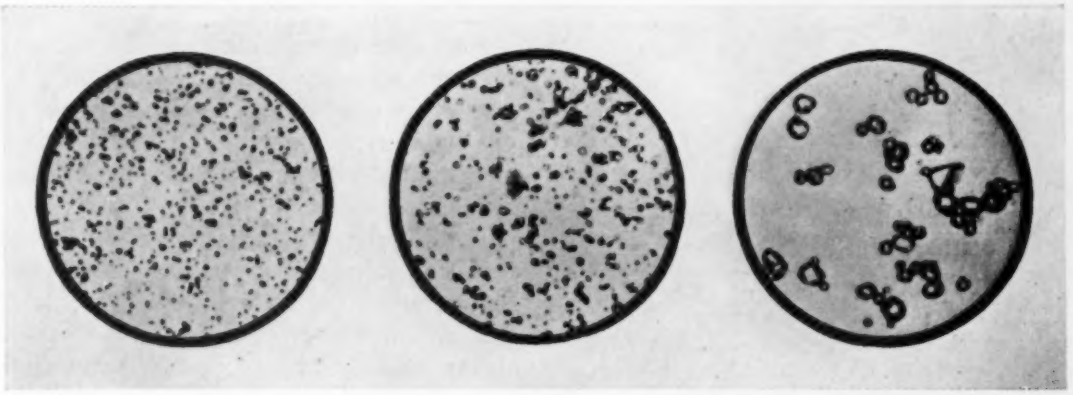
Another reason why intensifying screens decrease the sharpness of the roentgen-ray image is due to the fact that between screen and emulsion layer there is always a small air space, which even in cases of the most perfect physical contact between screen and emulsion is still great enough to cause spreading of the fluorescent light before entering the emulsion layer. This spreading of the fluorescent light from the

screen becomes especially noticeable when the intensifying screen in the cassette is not firmly pressed against the film.

A third factor contributes to the loss in detail sharpness due to the intensifying screens. It has just been mentioned that the tungstate grains should be as close as possible to the emulsion grains which they eventually will expose. This purpose could be achieved best by having the tungstate crystals all in one layer and this layer held in a perfect physical contact with the film. This condition, however, is not quite attainable, even if the layers of the screens are made very thin. There are always tungstate grains which are at different depths in the screen layer. Their distance from the emulsion layer is greater, and the fluorescent light, coming from those tungstate particles, is scattered and contributes to the loss in detail sharpness.

In spite of this disadvantage of the intensifying screens, the non-screen technique was not very frequently used, due to the lack of speed of the regular roentgen films for direct roentgen radiation. The high speed roentgen films were for a long time the only film types available and they had to serve two purposes, which from the standpoint of emulsion making could not very well be combined to greatest advantage in one emulsion.

The regular roentgen films had to have extreme high speed and high contrast for exposures with the fluorescent light of the intensifying screens. At the same time they should also be as sensitive as possible for direct roentgen-ray exposure. In combining these two features in one film, a certain compromise had to be made of both techniques. According to the relative importance, up to now, the direct roentgen-ray sensitivity had been gradually sacrificed in favor of improvements in sensitivity and gradation for the fluorescent light of intensifying screens.



A *B* *C*

Fig. 1. *A*, high speed roentgen-ray emulsion grain; *B*, detail screen; *C*, high speed screen. 250×.

It was felt, however, that the superiority of direct roentgen-ray images was too important to be sacrificed by the use of films which have been developed primarily for the intensifying screen technique. The problem of producing an emulsion which is especially well suited for direct roentgen radiation became more and more important.

Early in 1936 a special non-screen film was brought out to fill this existing need. As the name implies, this non-screen film was intended for use without intensifying screens. The emulsion was specially made to produce highest speed and greatest contrast for *direct* roentgen radiation. The sensitivity of this film to fluorescent light was rather low and this type therefore

Fig. 2. Roentgenogram of knee taken in posteroanterior position; non-screen film with Potter-Bucky diaphragm; 63 kv. (peak), 200 ma.

could not be used to advantage in connection with intensifying screens. This special non-screen film was very favorably received and revived a widespread interest in the non-screen roentgen technique. The sensitivity of this new film for *direct* roentgen radiation was about 100 per cent higher than that of the regular high speed roentgen films. While the higher speed of this film for non-screen technique was of course of advantage, the sensitivity was still not sufficient to enable practicable exposures of heavier parts of the body without screens.

The superiority of the non-screen film for certain purposes, however, was convincing proof that if the sensitivity of this film type could be sufficiently increased to make it also practical for heavier parts of the body, a distinct advancement in the direction of better roentgen-ray images with more detail and better definition could be achieved. This increase in speed was possible and a *new* non-screen film, first shown at the Fifth International Congress of Radiology was produced, having five and a half times higher sensitivity as compared to the regular roentgen film used without screens.

This increased sensitivity greatly extended the possibilities of non-screen roentgenography. It made possible non-screen roentgen exposures of heavier parts of the body, such as cervical and dorsal spine, hip, shoulder, sacrum, etc. Also chest exposures of individuals with chest thicknesses not exceeding 10 inches are possible without special equipment. The remarkable detail sharpness of this type of roentgenogram is especially striking and of greatly enhanced diagnostic value.

For individuals with chest thicknesses exceeding 10 inches the use of non-screen film is not yet advisable because high kilovoltage (peak) must be used and the resulting image is apt to be too flat. The limitations for roentgenograms of the trunk of the body using this new film are approximately 175–190 pounds for a man and 130–140 pounds in the case of a woman. These limitations in regard to the size and weight of the patients indicate that the new non-screen film does not, and is not intended to take the place of high speed roentgen films and intensifying screens.

There probably will always be a great number of subjects where the intensifying screen technique will be preferable; for instance, on all trunk exposures of heavy individuals or in cases where the subject normally shows very little differentiation in structure and contrast.

Due to the high sensitivity of non-screen film to primary roentgen radiation, the sensitivity for secondary radiation is also higher and the use of Potter-Bucky diaphragms, especially for heavier parts, is therefore recommended in order to obtain results of maximum quality. The use of proper Potter-Bucky diaphragms allows higher kilovolt (peak) values with greater penetration and the resulting images have

an appearance of transparency which is especially characteristic of this technique.

Details in bone canals become distinctly visible and even if bones are superimposed (see patella in Fig. 2) they are clearly outlined in their structure and relative position to each other.

The greater detail sharpness of non-screen roentgenograms makes possible another important advantage for the examination of these images. Without apparent loss in detail sharpness these roentgenograms can be viewed under low power magnification, which in many cases proves to be very helpful for diagnostic purposes. Minute lesions which are ordinarily not visualized or which are scarcely perceptible can be closely examined by the use of magnifying glasses, especially if small focal spot tubes have been used.

It would be of little practical value to examine roentgenograms, taken with intensifying screens, with magnifying glasses because, as pointed out earlier, the detail sharpness is not great enough to derive any benefit from magnification.

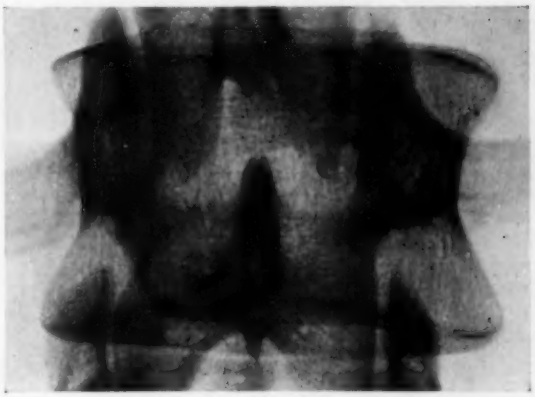
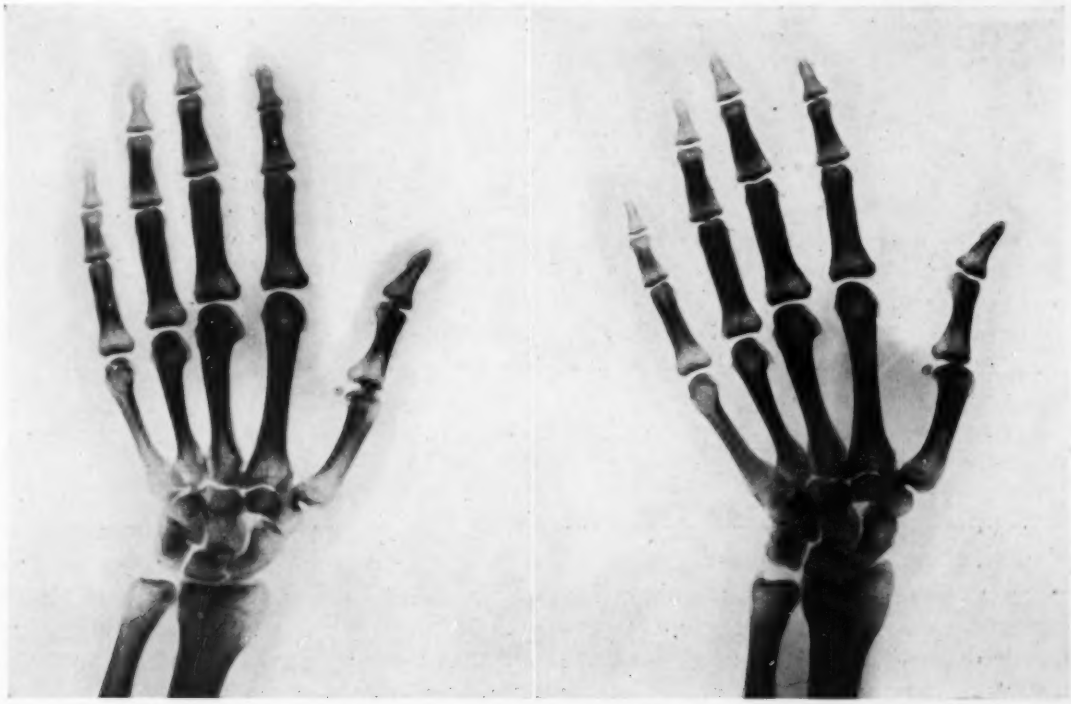


Fig. 3. Section of fourth lumbar vertebra enlarged about 3 diameters.

The non-screen technique in general allows a greater flexibility and latitude of



A *B*

Fig. 4. *A*, exposure on regular roentgen film with intensifying screen; 36 kv. (peak), 100 ma., 2/3 sec., 40-inch distance, no Potter-Bucky diaphragm. *B*, exposure on non-screen film; 36 kv., 100 ma., 4 sec., 40-inch distance, no Potter-Bucky diaphragm.

CHART I

TECHNIQUE FOR NEW NON-SCREEN FILM

Part	Position	Milli-amperes	Time sec.	Kilovolts (peak)	Distance	Potter-Bucky Diaphragm
Wrist	PA	100	2	36	40″	No
Elbow	AP	100	2	43	40″	No
Elbow	Lat.	100	2	45	40″	No
Foot	PA	100	2	43	40″	No
Ankle	Lat.	100	2	43	40″	No
Knee	Lat.	100	2	50	40″	No
Shoulder	AP	100	2	52	40″	No
Atlas and axis	AP	100	2	52	25″	No
Profile	Lat.	100	2	36	40″	No
Wrist	PA	100	2	50	40″	Yes
Shoulder	AP	100	2	65	40″	Yes
Knee	Lat.	100	2	63	40″	Yes
Ankle	Lat.	100	2	56	40″	Yes
Sacrum	AP	100	4	65–70	40″	Yes—cone
Hip	AP	100	4	65–70	40″	Yes—cone
Cervical spine	Lat.	100	5	70	48″	Yes—cone
Cervical spine	AP	100	3	70	48″	Yes—cone
Dorsal spine	Lat.	100	2	60–75	40″	Yes
Dorsal spine	AP	100	2	50–75	48″	Yes
Chest—10″	PA	40 M.A.S.		70	48″	No
Mastoid	Lat.	100	2	76	25″	Yes
Skull	Lat.	100	4	76–80	25″	Yes
Wrist	PA	100	2	44	40″	Yes
Wrist	PA	100	1	42	40″	No
Elbow	AP	100	1	49	40″	No
Elbow	AP	100	2	51	40″	Yes
Feet	Lat.	100	2	48	40″	Yes
Feet	Lat.	100	1	46	40″	No
Ankle	Lat.	100	1	51	40″	No
Ankle	Lat.	100	2	53	40″	Yes
Profile	Lat.	100	2	40	40″	Yes
Atlas and axis	AP	100	2	65	22″	Yes
Shoulder	AP	100	2	71	40″	Yes
Knee	Lat.	100	2	58	40″	Yes
Knee	AP	100	2	62	40″	Yes
Pelvis	AP	40	10	70	40″	Yes
Lumbar	AP	40	10	70	40″	Yes
Dorsal	AP	100	2	78	36″	Yes
Chest	PA	400	1/10	70	48″	No

exposure. This is especially true in using the special non-screen film. This film has a longer scale of densities and therefore shows a more correct response and a better intermediate tonal gradation.

In Figures 4*A* and 4*B* this point has been exemplified by making exposures of a hand on regular roentgen film with intensifying screen (Fig. 4*A*) and without screen on non-screen film (Fig. 4*B*).

The exposures have been made for a good penetration of the wrist and 36 kv. (peak) has been used in both cases. Even though the reproduction minimizes the differences somewhat, the non-screen image (Fig. 4*B*) shows the terminal phalanges much clearer

and more distinctly outlined, demonstrating the greater latitude of non-screen film and technique. In addition, the difference in detail sharpness between the two roentgenograms is very noticeable.

It is difficult to prescribe a non-screen technique based on this new film which will be acceptable to all roentgenologists, since there is such a divergence of opinion as to what constitutes the most satisfactory roentgenograms from a diagnostic standpoint. For this reason an exposure chart (Chart 1) is offered only as a basis from which the individual user may work out that technique which is best suited for his requirements.

The handling of the non-screen film is very similar to that of regular roentgen film, when used in cardboard holders. Each sheet of film lies between black interleaving paper which can be left around the film in the exposure holder as a precaution against possible light leakage through the cardboard holder. It is imperative that only lead backed holders be used in order to prevent fogging of the film by back-scattered radiation from the table top. It has been found advantageous to use the film in ordinary cassettes after the screens have been removed, because it is less difficult to place the cassettes properly, for instance in case the Potter-Bucky diaphragm is being used. There are also available special cassettes for non-screen films. For heavy parts like sacrum, hip or cervical or dorsal spine a cone or proper diaphragm should be used in order to minimize scattered radiation.

After the exposure has been made, the film is developed in the regular developer for about six minutes at or near 65°F.

Fig. 5. Anteroposterior roentgenogram of lumbar spine. Weight of patient 140 pounds. Non-screen film, 70 kv. (peak), 40 ma., 10 sec., 40-inch distance, Potter-Bucky diaphragm.

Fig. 6. Roentgenogram of shoulder taken with non-screen film; 70 kv. (peak), 200 ma., 40-inch distance, Potter-Bucky diaphragm.

Longer development up to eight minutes will produce greater contrast.

It is advisable to develop these films by time rather than by inspection. Similar to all roentgen films, the non-screen film should be thoroughly rinsed between development and fixing by frequent immersion and withdrawal in running water or in an acid rinse bath.

In conclusion, it may be said that this *new* non-screen film is a definite contribution to the roentgen art and places at the disposal of the profession a medium which has not only permitted a revival of non-screen technique but, due to the five and a half to six times higher speed, has made is possible to use this generally superior technique not only for extremity work, but also for most of the heavier parts of the body. The advantages to be derived from this film are manifold: maximum detail, better definition, wider exposure latitude and greater diagnostic value.

CASE REPORT FROM THE WEEKLY SEMINAR OF THE ROENTGENOLOGICAL DEPARTMENT OF THE MASSACHUSETTS HOSPITAL, BOSTON, MASSACHUSETTS

Prepared by JAMES R. LINGLEY, M.D.

A WHITE MALE, aged twenty, entered the hospital because of a tumor mass in the left thigh of ten months' duration. The mass had progressively increased in size during this time and recently a dull ache had been noticed in this region. There was no history of injury and the patient felt well except for slight pain. Physical examination was essentially negative except for the finding of a hard mass, about the size of a grapefruit, deep in the soft tissues of the left upper thigh. The skin was freely movable over it and it seemed to be attached to the bone. It was non-tender, non-fluctuant, and there was no increased temperature in the affected area. Roentgen examination of the chest was negative.

DISCUSSION

DR. HOLMES: The films on this case show a soft tissue tumor just below the greater trochanter. In the central portion of the tumor there is a calcium deposit which has no definite pattern (Fig. 1). The bones are of normal structure and density and there is no erosion. (Lateral views did not reveal additional information.) There is no tendency of the periosteum to spread out into the tumor. From the evidence we have, I would assume that this is a tumor arising in the soft tissues and in no way connected with the bone.

The film of the chest is entirely negative. We have then a soft tissue tumor in the center of which there is calcium. The first thing we should decide, of course, is whether it is an inflammatory process or a neoplasm. If inflammatory, we should consider tuberculosis. A cold abscess might contain calcium but such an abscess should have a source in the bone and I see no evidence of destruction of the bone anywhere and nothing to suggest a tuberculous focus. The calcium is not deposited in the way one would expect it to be if it were tuberculosis. Another thing to consider is calcifying hematoma. On the character of the calcium deposit, I cannot rule it out, but I would be very much against this diagnosis.

We are then forced to assume a neoplasm. The next question is whether it is benign or malignant. I do not know of any benign neoplasm that looks like this. Of the malignant

FIG. 1. Note the soft tissue mass with irregular calcium deposit in its central portion; also, the thickening of the cortex of the femur directly beneath it.

neoplasms that might give such a picture, the first one to think of is fibrosarcoma. Fibrosarcoma would arise from the fascia producing a large tumor, occurring at any age, although it is more common in older people and it could produce calcium in this way. Fibrosarcoma would seem to me to be the most reasonable diagnosis.

DR. HAMPTON: When you were discussing hematoma, did you think the duration had any bearing on the character of the calcification?

If it existed for ten months would the calcium be better organized or would it disappear at this time?

Dr. Holmes: I am not at all certain that I can answer definitely. There is a period of beginning calcification and one of disappearing calcification, but I have never seen one that appeared as this does.

Dr. Hampton: I wonder if the calcification would be flatter if it were being absorbed.

Dr. Lingley: At the Dillon Field House, Dr. Thorndike has had 8 cases of myositis ossificans among the Harvard University athletes. We have been able to follow these cases with periodic roentgen examination from the time of injury onward and have thus been able to determine the date of the first appearance of calcification and to follow its progress and regression. We have found that the calcification is first detectable quite uniformly three weeks after the injury and its progression is rapid from then onward. If this case were a calcifying hematoma, its age should be about four to five weeks and not ten months as the history states. At ten months, the calcification in myositis ossificans is a well-organized, dense mass with sharply defined smooth borders, quite unlike the discrete flecks present in this case.

Dr. Hampton: Then we have another question. We did not know whether or not the cortex of the femur was actually eroded a little.

Dr. Sosman: We had an osteogenic sarcoma almost in the center of a fibrosarcoma which looked very much like this. It produced actual bone. The shadows in this case are tiny punctate areas of calcification, not like bone structure. It does not have the laminated structure of calcified hematoma. It looks to me as if the femur is a little flat opposite the tumor. I would say that it is flattened by pressure rather than by destruction. This might very well be osteogenic sarcoma developing in fibrosarcoma.

Dr. Castleman, Pathologist: The surgeons removed the whole tumor intact and did not find any extension to the bones. Grossly the tumor measured about 15×10×5 cm. and upon cutting through it we were able to feel and hear the spicules of calcium throughout the tumor. Histologically those spicules are areas of calcium deposited in the tumor but without any bone formation. We have classified the tumor as fibrosarcoma. It is, however, an unusual type of fibrosarcoma. In many places the cells are short, undifferentiated and comma shaped and if the tumor were near a joint, we might call it a synovioma.

Dr. Lingley: The prognosis is rather poor, is it not?

Dr. Castleman: Yes. It appears to be growing very fast. If it is synovioma it is bound to recur.

Dr. Sosman: Was the tumor definitely demarcated?

Dr. Castleman: Yes.

Dr. Hampton: Was there any hemorrhage into it?

Dr. Castleman: No.

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Twenty-fourth Annual Meeting: 1939, to be announced.

EDITORIALS

THE ANNUAL MEETING

THE Thirty-ninth Annual Meeting of the American Roentgen Ray Society will be held at Haddon Hall, Atlantic City, September 20 to 23, 1938. The President-Elect, Dr. John W. Pierson, and his committee have arranged an excellent program, which is given in detail elsewhere in this issue of the JOURNAL. The advantages of Atlantic City as a meeting place are too well known to need further comment.

The annual meeting of the Society offers not only an opportunity to the members for an exchange of ideas with regard to the new investigations which they have conducted during the past year, but it also gives a splendid opportunity to see the new technical equipment which has been devised. This year there promises to be an unusually large display of equipment and of improvements and accessories in the Commercial Exhibit. The Scientific Exhibit being arranged by the committee of which Dr. Vincent W. Archer is Chairman also will be an extensive and important one and will be worth much study.

It is of particular interest that this year's meeting falls on the twenty-fifth anniversary of the announcement of two inventions which have had a far-reaching effect on radiology—the announcement of the Coolidge tube and of Potter's improvement of the Bucky diaphragm. These two additions to our armamentarium have advanced the technical side of roentgenology more than any inventions since the days of the Snook interrupterless transformer. One has only to stop and consider the tremendous advancements that have been made in the diagnosis of intracranial lesions, of lesions of the gastrointestinal and genitourinary tracts, and particularly of diseases and deformities of the spine, all of which have been made possible by the perfection of the Potter-Bucky diaphragm and the introduction of the Coolidge tube. Indeed, one may run the whole gamut of diagnostic roentgenology and find no place in which the Potter-Bucky diaphragm has not been of assistance. This and the Coolidge tube have advanced our methods of examination far beyond the point dreamed of in the early days of roentgenology. No longer does the "dead hand" of custom and authority interpose its weight against new ideas and discoveries in roentgen diagnostic and therapeutic measures. On the contrary, any advancement in these fields are welcomed by all branches of medicine. This is a far cry from the early days of roentgenology. One has only to recall the great and almost vehement antagonism to the use of the roentgen ray in the study of pulmonary tuberculosis, and yet today, by general consent, it stands first among the procedures in the diagnosis of this condition.

The Caldwell Lecture, one of the high points of the meeting, will be given this year by Dr. Joseph McFarland of Philadelphia. His subject "Keeping in Step with Science" is a provocative one.

The Program Committee has arranged a number of Educational Courses which will be given by a group of selected individuals who are authorities in their particular field. This in itself should create an unusual interest in the forthcoming meeting.

The approach of the Annual Meeting of the American Roentgen Ray Society provokes pleasant thoughts; we not only look forward to a splendid program but to pleasant associations with old friends. The usual social features of the meeting have been arranged. The Annual Golf Tournament this year assumes a new significance in that there will be competition for the Willis F. Manges Memorial Cup, the announcement of which is carried elsewhere in this issue.

Therefore it is hoped that advantage will be taken of the attractive program and meeting place by as many members and others interested in radiology as possible. It is unnecessary to call attention to the fact that it is desirable to make reservations for the meeting at Haddon Hall at an early date.

THE STATUS OF THE RADIOLOGIST IN HOSPITAL PRACTICE

RECENT developments indicate that a solution to the problems arising from hospital-radiologist relations may be rapidly forthcoming, thus putting an end to an unfortunate controversy which has existed for a number of years between organized medicine and the organized hospital world. The advent of hospital care insurance and the determination on the part of a few hospital boards to bolster decreasing endowment income with profits from medical services performed in the hospital has brought the question to a head within the last few years.

Both sides of the controversy have taken a definite stand for perhaps the first time and now with the question clearly defined there is greater opportunity for positive solution. In a series of resolutions adopted at the San Francisco session in June, the American Medical Association quite definitely voiced its attitude that the practice of medicine, in all its branches, belongs to physicians and that the tendency of some hospitals to exploit specialists in radiology, pathology, and anesthesia should be deplored. When the House of Delegates threatened hospitals who exploited the profession or the public with removal from the approved list of the Council on Medical Education and Hospitals, the American Hospital Association made known its attitude in a published statement of principle published in its July Journal.

Thus the issues are clearly drawn and opposing forces are lined up for a final decision that will, among other things, solve the question of relations between hospitals and radiologists. The American Medical Association states that, "It is unethical and therefore contrary to good public policy for hospitals to participate in the practice of medicine in order to obtain an income to finance other hospital activities or to attempt to lower the cost of special medical services by methods that impair the quality of the service or prevent the development of an improved service."

Other statements in the report of the Board of Trustees which were approved by the House of Delegates declare that, "Today action must be directed against the attempt of corporations and associations to practice medicine by employing nurses and technicians under the tacit supervision of salaried physicians. . . . To offer a lower cost service, hospital administrators and even some physicians have delegated many anesthesia, radiologic and pathologic duties to nurses and technicians. . . . The opportunities for physicians to establish desirable practices in these fields are becoming so limited that they turn to other types of practice. . . . The proper method of remuneration for the hospital and the physician should enable the hospital to provide adequate building, equipment and personnel without necessitating an attempt to control or to employ physicians."

Following the publication of this report the Board of Trustees of the American Hospital Association called a meeting and lost no time in making it known that they represented an entirely different point of view. In a set of principles adopted on June 18, they declared that, "The primary obligation of the hospital is to provide all the services necessary for the diagnosis, treatment, and rehabilitation of the patient. . . . The performance of diagnostic and therapeutic procedures by staff members constitutes the practice of medicine *in* hospitals. It is not the practice of medicine *by* hospitals."

The question: Shall medicine be practiced by private self-employed physicians

on a fee-for-service basis, or shall it be practiced by employees of a hospital corporation which shall dictate the income to be derived by the practitioner from his profession?

The American Medical Association clearly recognizes that if hospitals are permitted to take over the practice of radiology, pathology, and so forth, they are likely to take over other specialties as hospital practice increases. Said the report of the Board of Trustees, "The present situation in the practice of anesthesia, pathology, and radiology in hospitals and the potentiality of increasing the number and variety of special medical services controlled by hospitals call for the establishment of [an] ideal standard designed to permit a constant improvement of practice in hospitals."

New complications arose when the Philadelphia County Medical Society, after a two-year battle to exclude radiologic services from the hospital benefits offered in a proposed group insurance plan, sought an injunction to stop hospitals from proceeding with a plan which included certain medical services. Evidence is now being heard by a Master appointed by the Court who will submit his findings of fact to the Court. Representatives of organized hospitals have been asked to testify and it is understood that the Philadelphia County Medical Society has been promised assistance from the American Medical Association.

In an editorial in its July issue, the *Journal of the American Hospital Association* deplores the action taken in Philadelphia. Under the title, "We Take Our Stand," the Journal lamented that the positive stand may "compel hospitals generally to fight to maintain existing and necessary standards of organization and service."

Whatever the outcome, radiologists should be mindful and appreciative of the firm stand taken by the American Medical Association in support of principles long desired by radiologic organizations. The official pronouncements of the House of Delegates strongly support the contention of radiologists that, whatever method is used for reimbursing the hospital for its maintenance costs in the radiologic department, no substantial net profit from the professional services of the physician should go to the hospital.

Following consideration of resolutions introduced by Drs. E. H. Skinner, F. F. Borzell, and C. H. Goodrich at the San Francisco session, the Council on Medical Education and Hospitals submitted a report concerning the practice of medicine in hospitals by radiologists, pathologists, and anesthetists. Said the report: " . . . The Council believes that these problems are of vital concern to the medical profession; unwise decisions at this time may lead to consequences that would be disastrous to physicians and to the public alike, and that, therefore, a serious study should be made of existing relationships between hospitals and physicians practicing therein, especially in the departments of anesthesia, radiology, pathology and physical therapy, with a view to standardizing the relationship of these services to the hospital and, where necessary, of reaffirming the principles of ethics involved." The report was adopted by the House.

In a subsequent resolution the House of Delegates reiterated its disapproval of the inclusion of radiology, pathology, and so forth, in group hospitalization plans unless the benefits are specified as cash payments to the subscribers for the cost of these services. The House also adopted a report by the Judicial Council concerning renting of radium and stating that, " . . . the prescribing and directing of its use in the case of a patient whom the prescriber has not examined or seen is an unethical medical procedure."

The Inter-Society Committee owes much to Drs. E. H. Skinner and F. F. Borzell for earnest attention to radiologic questions during the House sessions. The helpful and cooperative attitude of the headquarters staff of the parent medical body, the American Medical Association, is enabling radiologists to gradually work out sound solutions to the many problems arising in hospital practice.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. B. Peirce, Royal Victoria Hospital, Montreal, Canada. Annual Meeting: Haddon Hall, Atlantic City, N.J., Sept. 20–23, 1938.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Dr. E. L. Jenkinson, 1439 S. Michigan Ave., Chicago, Ill. Annual meeting, 1939: To be announced.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: St. Louis, Mo., 1939.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N.Y. Annual meeting: Pittsburgh, Pa., Nov. 27–Dec. 2 1938.

RADIOLOGICAL SECTION, BALTIMORE CITY MEDICAL SOCIETY
Secretary, Dr. Howard H. Ashbury, Baltimore. Meets third Tuesday each month, September to May.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets twice annually in May and September. Special meetings may be called by the Chairman.

RADIOLOGICAL SECTION, LOS ANGELES CO. MED. SOC.
Secretary, Dr. M. L. Pindell, 678 S. Ferris Ave., Los Angeles, Calif. Meets on second Wednesday of each month at County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. E. Mendelson, 120 E. 19th St., Brooklyn, N. Y. Meets monthly on first Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph S. Gian-Franceschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months, place of meeting selected by the host.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. R. G. Willy, 1138 N. Leavitt. Meets second Thursday of each month October to May inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. Harry Hauser, 3395 Scranton Rd. Meetings are held at 6:30 P.M. at the Cleveland Chamber of Commerce Club rooms on the fourth Monday of each month from October to April, inclusive.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. E. A. Schmidt, 4200 E. 9th Ave., Denver, Colo. Meets third Tuesday of each month.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. J. H. Lucinian, 403 Huntington Bldg., Miami, Florida. Meetings held twice a year, May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. R. C. Pendergrass, Prather Clinic Bldg., Americus, Ga. Meets twice annually: in November and at annual meeting of Medical Association of Georgia in the spring.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. E. P. Halley, Decatur and Macon County Hospital, Decatur, Ill. Meetings held quarterly, time and place designated by president.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. C. C. Taylor, 23 E. Ohio St., Indianapolis, Ind. Meeting held the second Sunday in May annually.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. S. G. Schenck, 115 Eastern Parkway, Brooklyn, N.Y. Meets at Kings County Med. Soc. Bldg. on fourth Thursday, each month, October to May, 8:30 P.M.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. C. S. Davenport, St. Lawrence Hospital, Lansing. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. S. A. Morton, Columbia Hospital, Milwaukee, Wis. Meets monthly. Place of meeting named by the president.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. H. M. Weber, Mayo Clinic, Rochester, Minn.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets first Wednesday of each month, at 6 P.M., at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. E. C. Vogt, 300 Longwood Ave., Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. W. J. Marquis, 198 Clinton Ave, Newark, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place designated by the president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. R. D. Duckworth, 170 Maple Ave., White Plains, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year—January, May, November.

PACIFIC ROENTGEN CLUB
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport, Pa. Annual meeting, May, 1939. Exact time and place to be decided.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. B. R. Young, Temple University Hospital. Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. H. W. Jacox, 4800 Friendship Ave., Meetings held second Wednesday each month, 4:30 P.M., October to June at various hospitals.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. S. C. Davidson, 277 Alexander St., Meets on second Thursday from October to May, inclusive, 8 P.M., Rochester Academy of Medicine Building.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. W. K. Mueller, University Club Bldg. Meets fourth Wednesday of October, January, March and May, at a place designated by the president.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Fran-

*Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

cisco. Meets monthly on first Monday at 7:45 P.M., alternately at Toland Hall and Lane Hall.

South Carolina X-Ray Society
Secretary, Dr. R. B. Taft, 105 Rutledge Ave., Charleston. Meets at time and place of South Carolina Medical Assn.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg. Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. G. D. Carlson, 3121 Bryan St., Dallas, Texas. Next regular meeting in San Antonio, Oct. 22, 1938.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. V. W. Archer, University Hospital, University, Va. Meets annually in October.

Cuba

Sociedad Cubana de Radiologia y Fisioterapia
Secretary, Dr. Francisco Padron, Enrique, Villuendas 64, Havana, Cuba. Meets monthly in Havana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets on the third Thursday of each month, from November to June inclusive, at 8:15 P.M., at 32 Welbeck St., London, W. 1, or as advertised.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:15 P.M. at the Royal Society of Medicine, 1, Wimpole St., London, W. 1.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to Nov. incl. for scientific discussion.

Canadian Association of Radiologists
Secretary, Dr. A. C. Singleton, Medical Arts Bldg., Toronto, 5, Ontario.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext., Halifax, N.S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

Continental Europe

Belgian Society of Roentgenology
Secretary, Dr. J. Boine, Avenue des Alliés, 134, Louvain (Belgium).
Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7, Madrid, Spain. Meets monthly in Madrid.

Société de Radiologie Médicale de France
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean, La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel.
Meets annually in different cities.

Société Francaise d'Electrothérapie et de Radiologie Médicale
Meets monthly on fourth Tuesday, except during month of August and September, 12 Rue de Seine, Paris.

Association of German Roentgenologists and Radiologists in Czecho-Slovakia
Secretary, Dr. Walter Altschul, German University, Prague, 11.52.

Deutsche Röntgen-Gesellschaft (Gesellschaft für Röntgenkunde und Strahlenforschung)
Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aerzte.
Permanent Secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

Süd- und Westdeutsche Röntgensellschaft
Meets annually in different cities.

Nord- und Ostdeutsche Röntgengesellschaft
Meets annually in different cities.

Dutch Society of Electrology and Roentgenology
Holds two meetings a year in Amsterdam, one in the Spring, and one in the fall.

Societa Italiana Radiologia Medica
Secretary, M. Ponzio, University of Turin, Prof. Turin

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania.
Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson, Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin.
Meets monthly, first Monday at 8 o'clock State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky.
Meets monthly on the first Monday at 8 o'clock, the place of meeting being selected by the Society.

Polish Society of Radiology
Secretary, Dr. Jan Kochanowski, 45 Gornoslazka St., Warsaw. Meets annually.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. B. Krynski, 11 Zielna St.
Meets once a month except in the summertime.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summertime:

Society of Medical Radiology in Sweden
Meets in Stockholm.

Society of Medical Radiology in Norway
Meets in Oslo.

Society of Medical Radiology in Denmark
Secretary, Dr. G. Biering, Copenhagen.
Meets the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

Society of Medical Radiology in Finland
Meets in Helsingfors.

Vienna Roentgen Society
Meets first Wednesday of each month, at 6:30 P.M. at Zentral-Röntgen Institut des allgemeinen Krankenhauses Alserstrasse 4.

Orient

Japan X-Ray Association
c/o Orthopedic Surgery, Tokyo Imperial University. Meets annually in April.

Kinki Roentgen-Abend Society
Director, Dr. Prof. Taiga Saito, Ogawaoike Tyoto Japan. Meets bi-monthly on third Sunday.

SCIENTIFIC PROGRAM OF THE THIRTY-NINTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

September 20 to 23, 1938

Haddon Hall, Atlantic City, New Jersey

Tuesday Morning, September 20, 1938

9:00 A.M.

Call to order, B. R. Kirklin, M.D., President.
Presentation of medals and certificates.
Installation of incoming President.
President's Address. John W. Pierson, M.D.

1. Radium treatment of Peyronie's disease. Robert E. Fricke, M.D., Rochester, Minnesota (15 min).
   Discussion (5 min.) to be opened by Charles A. Waters, M.D., Baltimore, Maryland.
2. Deep roentgen therapy of mammary carcinoma. William A. Evans, M.D., and Traian Leucutia, M.D., Detroit, Michigan (15 min.).
   Discussion (5 min.) to be opened by Edwin C. Ernst, M.D., St. Louis, Missouri.
3. Roentgen therapy of cancer in the buccal cavity and of the cervix uteri. Edwin A. Merritt, M.D., R. R. Rathbone, M.D. (by invitation), and Ralph M. Caulk, M.D. (by invitation), Washington, D. C. (15 min.).
4. Direct application of roentgen rays to cervix by means of speculum with expanding blades. Arthur W. Erskine, M.D., Cedar Rapids, Iowa (5 min.).
   Discussion (5 min.) of papers by Drs. Merritt, Rathbone and Caulk and Erskine to be opened by Bernard P. Widmann, M.D., Philadelphia, Pennsylvania.
5. Treatment of metastatic cervical lymph nodes. Charles L. Martin, M.D., Dallas, Texas (15 min.).
   Discussion (5 min.) to be opened by Douglas Quick, M.D., New York City.
6. Roentgen therapy of hygroma cysticum colli in children. Fred M. Hodges, M.D., L. O. Snead, M.D., and R. A. Berger, M.D. (by invitation), Richmond, Virginia (15 min.).
   Discussion (5 min.) to be opened by Eugene T. Leddy, M.D., Rochester, Minnesota.

11:30 Adjournment to inspect Scientific Exhibits.

1:00 P.M. Educational Courses.

Tuesday Afternoon, September 20, 1938

2:00 P.M.

1. Carcinoma as a complication of achalasia of the esophagus. Karl Kornblum, M.D., and Lawrence C. Fisher, M. D. (by invitation), Philadelphia, Pennsylvania (15 min.).
   Discussion (5 min.) to be opened by E. Forrest Merrill, M.D., New York City.
2. Roentgenologic disclosure and diagnosis of cancer of the cardia. B. R. Kirklin, M.D., Rochester, Minnesota (15 min.).
   Discusser to be announced.
3. Lymphosarcoma of the stomach: diagnosis and treatment. Vincent W. Archer, M.D., University, Virginia (15 min.).
   Discussion (5 min.) to be opened by Robert J. Reeves, M.D., Durham, North Carolina.
4. The relation of meal temperature to gastric motility and secretion. J. Gershon-Cohen, M.D. (by invitation), Harry Shay, M.D. (by invitation), and S. S. Fels, M.D. (by invitation), Philadelphia, Pennsylvania (15 min.).
   Discussion (5 min.) to be opened by T. Grier Miller, M.D. (by invitation), Philadelphia, Pennsylvania.
5. The roentgen diagnosis of idiopathic steatorrhea and allied conditions: practical value of the "moulage sign." John L. Kantor, M.D., New York City (15 min.).
   Discussion (5 min.) to be opened by Ross Golden, M.D., New York City.
6. The use of the Miller-Abbott tube in roentgen diagnosis. J. E. Lofstrom, M.D. (by invitation), and R. J. Noer, M.D. (by invitation), Detroit, Michigan (15 min.)
   Discusser to be announced.

4:30 P.M. Executive Session.

Tuesday Evening, September 20, 1938

8:30 P.M. Caldwell Lecture

Introductory Remarks. Leopold Jaches, M.D., New York City.

Caldwell Lecture. "Keeping in Step With Science." Joseph McFarland, M.D., Philadelphia, Pennsylvania.

Wednesday Morning, September 21, 1938

8:00 A.M. Educational Courses.

9:15 A.M.

1. Thoughts on paradoxical movement of the diaphragm and mediastinal shift. Cinefluorographic studies. William H. Stewart, M.D., and Francis H. Ghiselin, M.D. (by invitation), New York City (15 min.).
   Discussion (5 min.) to be opened by John T. Farrell, Jr., M.D., Philadelphia, Pennsylvania.
2. Correlation of post-mortem chest teleroentgenograms with autopsy findings. Benjamin Castleman, M.D. (by invitation), and Aubrey O. Hampton, M.D. (by invitation), Boston, Massachusetts (15 min.).
   Discussion (5 min.) to be opened by H. Dabney Kerr, M.D., Iowa City, Iowa.
3. Roentgen visualization of the soft tissues in pregnancy. William Snow, M.D., and Meyer Rosensohn, M.D. (by invitation), New York City (15 min.).
   Discussion (5 min.) to be opened by John R. Carty, M.D., New York City.
4. Internal dimensions of the female pelvis. Robert P. Ball, M.D., New York City (15 min.).
   Discussion (5 min.) to be opened by A. Louis Dippel, M.D. (by invitation), Baltimore, Maryland.
5. The anatomic and roentgenologic consideration of anterior and posterior notching of the vertebral bodies as seen in the new-born. Eugene P. Pendergrass, M.D., and George Wagoner, M.D. (by invitation), Philadelphia, Pennsylvania (15 min.).
   Discusser to be announced.

11:30 Adjournment to inspect Scientific Exhibits.

Wednesday Afternoon, September 21, 1938

2:00 P.M.

Symposium on Heart Disease. Arranged by Merrill C. Sosman, M.D.

1. Normal anatomy and estimation of heart size. Fred Jenner Hodges, M.D., Ann Arbor, Michigan.
2. The clinical value of visualization of the heart chambers, the pulmonary circulation and the great blood vessels in man. George P. Robb, M.D. (by invitation), and Israel Steinberg, M.D. (by invitation), New York City.
3. Pericarditis. Eugene Freedman, M.D. (by invitation), Cleveland, Ohio.
4. Acquired valvular heart disease. Merrill C. Sosman, M.D., Boston, Massachusetts.
5. Non-valvular heart disease. George Levene, M.D., Boston, Massachusetts.
6. Congenital heart disease. Hugo Roesler, M.D. (by invitation), Philadelphia, Pennsylvania.
7. Aorta and great vessels. Marcy L. Sussman, M.D. (by invitation), New York City.
   Discussion to be opened by Samuel Levine, M.D. (by invitation), and George W. Holmes, M.D., Boston Massachusetts.

Thursday Morning, September 22, 1938

8:00 A.M. Educational Courses.

9:15 A.M.

1. The present status of roentgen treatment of sinus infections. Whitmer B. Firor, M.D., and Charles A. Waters, M.D., Baltimore, Maryland (15 min.).
   Discussion (5 min.) to be opened by Fred M. Hodges, M.D., Richmond, Virginia.
2. The combined roentgen-ray and radium treatment of carcinoma of the cervix. Richard Dresser, M.D., Joe Vincent Meigs, M.D. (by invitation), and J. C. Rude, M.D. (by invitation), Boston, Massachusetts (15 min.).
   Discussion (5 min.) to be opened by William P. Healy, M.D. (by invitation), New York City.
3. Carcinoma of the anus: its incidence, pathology and radiological treatment. Ira I. Kaplan, M.D., and Sidney Rubenfeld, M.D. (by invitation), New York City (15 min.).
   Discussion (5 min.) to be opened by Robert E. Fricke, M.D., Rochester, Minnesota.
4. Skin reactions with 400 kv. and 200 kv. unfiltered radiation, illustrated with color photographs. John T. Murphy, M.D., and Clarence E. Hufford, M.D., Toledo, Ohio (15 min.).
   Discussion to be opened by Harold W. Jacox, M.D., Pittsburgh, Pennsylvania (5 min.), and Edith H. Quimby, Ph.D., New York City.
5. Roentgen-ray diffraction studies of human nervous tissue. Lawrence Reynolds, M.D.,

Henrietta S. Hayden, Ph.D. (by invitation), and Kenneth E. Corrigan, Ph.D. (by invitation), Detroit, Michigan (15 min.).

Discussion (5 min.) to be opened by William Bale, M.D. (by invitation), Rochester, New York.

11:30 A.M. Adjournment to inspect Scientific Exhibits.

Thursday Afternoon, September 22, 1938

2:00 P.M.

Symposium on Growth. Arranged by Eugene P. Pendergrass, M.D., Philadelphia, Pennsylvania.

1. Chemical structure and its relation to growth and development. Frederick S. Hammett, Ph.D., Philadelphia, Pennsylvania (by invitation).
2. Growth hormones in plants. Jacob R. Schramm, Ph.D., Philadelphia, Pennsylvania (by invitation).
3. Radiation, mutation and the gene. P. W. Whiting, Ph.D., Philadelphia, Pennsylvania (by invitation).
4. Viruses and abnormal growths. Baldwin Lucke, M.D., Philadelphia, Pennsylvania (by invitation).
5. Biology of the cancer cell. Stanley P. Reimann, M.D., Philadelphia, Pennsylvania (by invitation).

   Discussion to be opened by Stanley P. Reimann, M.D., Philadelphia, Pennsylvania (by invitation).

4:30 P.M. Executive Session.

7:00 P.M. Banquet.

Friday Morning, September 23, 1938

8:00 A.M. Educational Courses.

9:15 A.M.

1. Mediastinal glandular tuberculosis in the adult resembling Hodgkin's disease. George E. Pfahler, M.D., Philadelphia, Pennsylvania (15 min.).

   Discussion to be opened by Lloyd Freeman Craver, M.D., New York City (by invitation) (5 min.).
2. Liposarcoma of the lung and mediastinum. Charles W. Perkins, M.D., Norwalk, Connecticut (15 min.).

   Discussion (5 min.) to be opened by Ralph F. Bowers, M.D., New York City (by invitation).
3. Tuberculous tracheobronchitis. Carleton B. Peirce, M.D., Montreal, Canada (15 min.).

   Discussion (5 min.) to be opened by Charles R. Austrian, M.D., Baltimore, Maryland (by invitation).
4. Roentgen localization of acute lung abscess with particular reference to the oblique view. Coleman B. Rabin, M.D., New York City (by invitation) (15 min.).

   Discussion (5 min.) to be opened by Carleton B. Peirce, M.D., Montreal, Canada.
5. Methods for determining pineal position with analysis of their errors. Walter W. Fray, M.D., Rochester, New York (15 min.).

   Discusser to be announced.
6. Roentgen findings in renal tuberculosis. Henry K. Taylor, M.D., New York City (15 min.).

   Discussion to be opened by Bernard H. Nichols, M.D., Cleveland, Ohio (5 min.).
7. Lateral pyelography. E. L. Shiflett, M.D., and D. Y. Keith, M.D., Louisville, Kentucky (15 min.).

   Discussion (5 min.) to be opened by Bernard H. Nichols, M.D., Cleveland, Ohio.

11:30 A.M. Adjournment to inspect Scientific Exhibits.

Friday Afternoon, September 23, 1938

2:00 P.M.

1. Intracranial complications attending roentgen therapy for carcinoma of the scalp. Robert A. Groff, M.D. (by invitation), and Philip J. Hodes, M.D. (by invitation), Philadelphia, Pennsylvania (15 min.).

   Discussion (5 min.) to be opened by Cornelius G. Dyke, M.D., New York City.
2. The use of concrete protection for x-ray voltages up to 400 kv. Lauriston S. Taylor, Ph.D., and George Singer, Ph.D. (by invitation), Washington, D. C. (15 min.).

   Discussion (5 min.) to be opened by Otto Glasser, Ph.D. Cleveland, Ohio, and G. Failla, Ph.D., New York City.
3. Radium quantity meter. Robert B. Taft, M.D., Charleston, South Carolina (15 min.).

   Discussion (5 min.) to be opened by George E. Pfahler, M.D., Philadelphia, Pennsylvania.
4. Relief of pain. Francis C. Grant, M.D.,

Philadelphia, Pennsylvania (by invitation) (15 min.).

Discussion (5 min.) to be opened by Eugene P. Pendergrass, M.D., Philadelphia, Pennsylvania.

5. Aspiration biopsy of tumors in inaccessible organs under fluoroscopic guidance. John V. Blady, M.D., New York City (by invitation) (15 min.).

   Discussion (5 min.) to be opened by Hayes E. Martin, M.D., New York City (by invitation).

6. A method for obtaining greater ratio of deep to surface dosage: a preliminary report. Sydney J. Hawley, M.D., Danville, Pennsylvania (15 min.).

   Discussion (5 min.) to be opened by G. Failla, Ph.D., New York, and Otto Glasser, Ph.D., Cleveland, Ohio.

7. Roentgen treatment of inoperable carcinoma of the breast by multiple converging beams. Eugene T. Leddy, M.D., Rochester, Minnesota (15 min.).

   Discussion (5 min.) to be opened by G. Failla, Ph.D., New York, and Otto Glasser, Ph.D., Cleveland, Ohio.

Final Adjournment

EDUCATIONAL COURSES

The educational courses met with such universal approval at the Fifth International Congress of Radiology, that the program Committee deemed it advisable to introduce a series of courses in this year's program. Various members and invited guests of the Society have consented to conduct the courses, which will be given each day from Tuesday to Friday, inclusive. They will be open to visitors as well as to members of the Society. Anyone desiring to take the courses, whether member or visitor, will be required to register. Admission will be by card only.

The following is an outline of the program:

Tuesday, September 20, 1938
1:00 to 2:00 P.M.

Physics. Edith H. Quimby, Ph.D., New York City, and J. L. Weatherwax, Ph.D., Philadelphia, Pennsylvania.

Radiation Therapy of Lymphoblastomata. Edward L. Jenkinson, M.D., Chicago, Illinois.

Diagnosis of Chronic Pulmonary Lesions—Non-Tuberculous. LeRoy Sante, M.D., St. Louis, Missouri.

Diagnosis of Bone Changes in Infants and Children. Ralph S. Bromer, M.D., Bryn Mawr. Pennsylvania.

Wednesday, September 21, 1938
8:00 to 9:00 A.M.

Physics. J. L. Weatherwax, Ph.D., and Edith H. Quimby, Ph.D.

Radiation Therapy of Superficial Lesions. Bernard P. Widmann, M.D., Philadelphia, Pennsylvania.

Diagnosis of Kidney Tumors. Charles A. Waters, M.D., Baltimore, Maryland.

Diagnosis of Intracranial Lesions. Cornelius G. Dyke, M.D., New York City.

Thursday, September 22, 1938
8:00 to 9:00 A.M.

Physics, Edith H. Quimby, Ph.D., and J. L. Weatherwax, Ph.D.

Radiation Therapy of Carcinoma of the Breast. U. V. Portmann, M.D., Cleveland, Ohio.

Diagnosis of Bone Tumors. Charles F. Geschickter, M.D., Baltimore, Maryland (by invitation).

Diagnosis. Body Section Roentgenography. Sherwood Moore, M.D., St. Louis, Missouri.

Friday, September 23, 1938
8:00 to 9:00 A.M.

Physics. J. L. Weatherwax, Ph.D., and Edith H. Quimby, Ph.D.

Radiation Therapy of Uterine Carcinoma. Charles L. Martin, M.D., Dallas, Texas.

Diagnosis of Bone Tumors, Charles F. Geschickter, M.D., Baltimore, Maryland.

Diagnosis of Gastrointestinal Lesions. B. R. Kirklin, M.D., Rochester, Minnesota.

THE ANNUAL GOLF TOURNAMENT

The Committee has inaugurated a new feature of the annual golf tournament in the awarding of the Willis F. Manges Memorial Cup. The inauguration of this feature of the annual golf tournament was born of a desire on the part of the friends of Willis Manges to do honor to him and to perpetuate his memory. His keen zest for the

game of golf, in which he excelled, was well known, and the awarding of a cup named in his honor seemed quite appropriate. This cup has been obtained as the result of popular subscription among the members of the Society. It is to be put into competition on September 19, 1938. This event will be held annually and will serve to remind us of Willis Manges, whom we all knew as a great radiologist and as a master golfer.

RENTAL OF RADIUM

A widespread practice of renting radium for the treatment of patients by physicians not owning or being experienced in the use of radium has caused considerable discussion during the past year. Ordinarily instructions in the technic of the use of radium are sent by the person furnishing it. Sometimes the radium is furnished by a commercial concern, sometimes by a physician owning it. The advisability of the use of such a powerful agency by those not trained in its use and the ethics involved of prescribing and directing its use by a person who has not examined or seen the person on whom it is to be used has come before the Council. As a result of a rather extensive correspondence both from those favoring its use as described and those opposed, the Judicial Council is of the opinion that the prescribing and directing of its use in the case of a patient whom the prescriber has not examined or seen is an unethical medical procedure. The Council recognizes that advice and help in difficult cases is often furnished by those in a position to be of possible or probable assistance but it believes that the great dangers accompanying the use of radium removes that particular remedy from the field of advice without personal contact with the patient.*

The Judicial Council of the American Medical Association is responsible for the above report. It was presented to the House of Delegates of the American Medical Association at the 1938 San Franciso session. The Reference Committee of the House reported back as follows:†

Rental of Radium: There was an objection to the last sentence in that paragraph pertaining to rental of radium, which the committee considered most carefully. After due consideration of the matter, your committee feels that the phraseology should remain. Any exception to the general principle involved would defeat its purpose.

The House adopted this report unanimously.

This report and result is brought forth after several years of effort upon the part of the American Radium Society, American Roentgen Ray Society and the Radiological Society of North America. The original resolutions, as proposed by the American Radium Society and concurred in by the unanimous action of the Councils and memberships of the other two radiological societies were as follows:

Resolutions adopted by the American Radium Society at Annual Meeting, Cleveland, June 12, 1934:

Whereas, it is now recognized that radium has been demonstrated to be of definite value in the treatment of disease, and

Whereas, some states and many communities in the country have little or no radium available, and

Whereas, funds are not always available for the purchase of suitable preparations of radium for use by those physicians who are qualified in radium therapy, and

Be It Resolved, that we recommend the refusal of advertising matter in national and state journals when the companies concerned are advertising Medical Consulting Service or are advertising such service through the mails in connection with their sale or rental of radium.

Be It Resolved, that we disapprove of any doctor's acting as a consultant to a commercial company carrying on such a campaign of advertising, and that we consider such an association sufficient grounds to warrant withholding the certificate of the American Board of Radiology.

Be It Resolved, that we recognize the ethical commercial companies as a necessity. It is the advertised consulting service which is at fault and which should be condemned. It is recognized that such restrictions on the advertising of a medical service will in no way hamper properly qualified radium therapists in obtaining adequate supplies of radium or radon

* *J. Am. M. Ass.*, April 30, 1938, *110*, 1489.
† *J. Am. M. Ass.* July 2, 1938, *111*, 47.

for the purposes in which they are qualified to use it.

Be It Resolved, that we approve an informal medical consultant for the guidance of those commercial companies which refrain from advertising such professional service either publicly or privately, and that in such case their informal consultant be one approved by the American Board of Radiology.

Be It Resolved, that the same criticism be applied to many individual owners of radium.

Be It Resolved, that we regard the approval of the American Board of Radiology as the best standard of qualification for those assuming the responsibility of using radium. But we recommend as wide publicity of this Board's existence and approval as is possible to the public, consistent with ethical practices, as the most effective safeguard which can be afforded them.

This action of the Judicial Council and the House of Delegates of the American Medical Association is welcomed and enjoys hearty endorsement by our various radiological societies. The American Radium Society recognized the merit of this action and passed a motion to write a letter of appreciation to the Chairman of the Judicial Council, Dr. George E. Follansbee of Cleveland.

In the discussion before the House of Delegates it was brought out that this resolution did not prevent the sale, rental, leasing or borrowing of radium element or emanation but it did oppose the mail order consultation of a physician upon the meager description of a case by a physician. It opposes the prescription or consultation by mail and demands that a consultant see, examine and plan the radium treatment only by personal contact with the patient. The report implies that it is for the best interests of the public and the profession to insist that radium therapy be applied by those physicians who know how to use radium and who know how and what dosage and method of radium therapy will serve the patient best. It is not essential that a physician applying radium therapy be a radiologist or that he possess or own the radium used. He may rent, buy, borrow or lease the radium or the emanation. He may not be ignorant of the principles of radium practice and methods and depend upon the advice of a distant consultant who has never seen the given case for the radium element, emanation, apparatus or dosage. Mail order radium treatment will no longer be an ethical and proper procedure.

This report and action of the Judicial Council and the House of Delegates of the American Medical Association is not a means of reserving radium therapy to radiologists or radium specialists and by no connotation can this action be inferred as prejudicial to the application of radium in the treatment of patients by any physician who by study and experience is capable of conducting such treatment. It does specifically insist that no corporation can indulge in the practice of medicine so far as employing or even informally asking a physician, radiologist or otherwise, to consult upon the apparatus, method and dosage of radium element or emanation by mail, upon the description by letter or telegram of a patient's condition. There is no reason why a company, possessing radium element or emanation, should not continue to sell its products to physicians upon order from a physician but it cannot employ a physician or radiologist to direct, advise, consult or design its products without a personal examination of the given patient. This does not prevent any physician, be he radiologist or otherwise, from allowing his radium possessions to be used upon other patients than his own but he must not estimate the dosage or the method of radium application upon other than the information he gathers from an examination of the given patient.

This definition of the ethical aspects of radium therapy is timely. In fact it is overtimely. When the first resolutions were presented by the radiological societies, there was some misgiving as to whether or not it was a means of preserving radium therapy to radiologists. There was the accusation that there might be some measure

of self-preservation in the resolutions. Possibly that is one reason for the careful wording of the report of the Judicial Council after many months of deliberation and due consideration for the motives and ideals involved. Public policy and medical ethics are served in the highest degree by this excellent report.

E. H. Skinner
Delegate, Section of Radiology
American Medical Association

INTERESTING NEWS ITEMS FOR RADIOLOGISTS

The meeting of the American Medical Association at San Francisco June 13 to 17, 1938, provided many reports and resolutions in the proceedings of the House of Delegates and the various councils and reference committees. There are now several radiologists within the membership of the 173 delegates from the states and sections of the American Medical Association. Their geographical distribution makes it possible to obtain a spread of interest and establish liaison among all of the states' delegates. A radiologist, Dr. Roy W. Fouts, of Omaha, Nebraska, was elected Vice-Speaker of the House of Delegates.

The items of interest for radiologists in the various proceedings were principally involved in the discussions upon the physician-hospital relationships, and the practice of specialists in hospitals; the employment of physicians, surgeons, radiologists, pathologists and anesthetists by hospitals. All of these propositions involved the ethics of medical practice and the question as to whether a corporation can employ physicians in any capacity, where the hospital derives profit from fees collected by the hospital for the services of such physicians above and beyond the costs of such services. The question as to the possible division of medical practice into technical and professional services and the all-inclusive payment plan for hospital and professional services received some emphasis.

The report of the Judicial Council and the approval of the report by the House of Delegates upon Radium Rental have been discussed in another item in this Journal.

The Resolutions upon which the discussions were based were presented by two radiologists and are here copied verbatim from the Proceedings of the House of Delegates.*

Resolutions Requesting the Council on Medical Education and Hospitals to Study the Status of Clinical and Pathologic Laboratories and Other Departments in Hospitals

Dr. Francis F. Borzell, Pennsylvania, Presented the following resolutions, which were referred to the Council on Medical Education and Hospitals:

Whereas, The American Medical Association has through its House of Delegates and its various bureaus and councils from time to time propounded certain principles of ethics and established standards of relationship looking toward the maintenance of the highest quality of professional service both in private practice and in hospitals; and

Whereas, Many of these pronouncements refer specifically to those special branches of medicine involving somewhat unusual relationship between hospitals, institutions and the physician practicing these specialities in the hospital; and

Whereas, Certain usages have sprung up which are tending to involve ethical and legal considerations; and

Whereas, It would appear to be highly desirable and in the best interests of good medical practice and sound public policy to have certain of the aforementioned pronouncements clarified, codified and in some instances reiterated; be it

Resolved, That the Council on Medical Education and Hospitals be requested to study the status of clinical and pathologic laboratories, radiologic departments and physical therapy departments in hospitals and institutions with a view toward standardizing the relationship of these services to these institutions and when necessary reaffirming principles of ethics involved in these relationships; and be it further

Resolved, That the Council be authorized to confer with such agencies as are deemed necessary to arrive at these conclusions.

Resolution on Standards for the Practice of Medicine in Hospitals

Dr. E. H. Skinner, Section on Radiology, presented the following resolution, which was re-

* *J. Am. M. Ass.*, July 2, 1938, *111*, 39; 41; 45; 62.

ferred to the Council on Medical Education and Hospitals:

WHEREAS, The Board of Trustees of the American Medical Association, in its annual report, has directed attention to certain conditions in these words, "The present situation in the practice of anesthesia, pathology and radiology in hospitals and the potentiality of increasing the number and variety of special medical services controlled by hospitals call for the establishment of a similar ideal standard designed to permit a constant improvement of practice in hospitals"; and

WHEREAS, The said Board in its report has further pointed out that the turmoil between hospitals and national societies of anesthetists, of pathologists and of radiologists cannot continue without harmful effects on the practice of medicine in hospitals; be it

Resolved, That this House of Delegates instruct its Board of Trustees to direct the Council on Medical Education and Hospitals to proceed toward the creation of standards for the practice of medicine in hospitals, such standards to include the requirements for approval of hospitals and the standards which govern the relations between physicians and hospitals.

Report of Reference Committee on Medical Education

Dr. William D. Johnson, Chairman, presented the following report:

1. Resolutions on Medical Practice in Hospitals: The resolutions introduced by Dr. E. H. Skinner, Section on Radiology, Dr. Francis F. Borzell, Pennsylvania, and Dr. Charles H. Goodrich, New York, were taken up and given consideration by the Council on Medical Education and Hospitals acting as a reference committee and will be reported on by that body.

2. Resolutions Requiring of Foreign Graduates Full Citizenship in the United States: The resolutions introduced by Dr. William R. Molony Sr., which in essence express the desirability that foreigners, graduates of foreign institutions, be required to obtain full citizenship in the United States of America before being admitted to practice, meet the full approval of your committee.

3. Report of Council on Medical Education and Hospitals: Your committee has given due and proper consideration to the report of the Council on Medical Education and Hospitals and finds itself in accord with the several recommendations of the Council.

Your reference committee particularly wishes to commend the survey of medical education made by the Council and believes that this study should be so published as to constitute a permanent record of the Association's contribution to this field of professional education. If further funds are required for the final consummation of this project, the committee recommends that proper appropriations be made for this purpose.

Your committee wishes to endorse the report of the Council on Medical Education and Hospitals as a whole, including Supplement A stating the essentials of an acceptable medical school

Respectfully submitted.

WILLIAM D. JOHNSON, Chairman.
JOHN W. AMESSE.
J. C. FLIPPIN.
MCLAIN ROGERS.
WALTER E. VEST.

On motions, duly seconded and carried, the report of the Reference Committee on Medical Education was adopted section by section and as a whole.

Report of Council on Medical Education and Hospitals

Dr. W. D. Cutter, Secretary of the Council, presented the following report of the Council on Medical Education and Hospitals acting as a reference committee:

The resolutions of Dr. Francis F. Borzell, Pennsylvania, Dr. Charles H. Goodrich, New York, and Dr. E. H. Skinner, Section on Radiology, which were referred to the Council on Medical Education and Hospitals, all dealing more or less with the same subject, were considered together. The proposers of these resolutions, the delegates from the Massachusetts Medical Society, members of the California Medical Association and others met with the Council to express their views concerning the problems that concern the practice of medicine in hospitals by radiologists, pathologists and anesthetists. These problems have been rendered more acute by the rapid extension of systems of group hospital insurance within the last few years. The Council believes that these problems are of vital concern to the medical profession; that unwise decisions at this time may lead to consequences that would be disastrous to physicians and to the public alike, and that, therefore, a serious study should be made of existing relationships between hospitals and the physicians practicing therein, especially in the departments of anesthesia, radiology, pathology and physical therapy, with a view to standardizing the relationship of these services

to the hospital and, where necessary, of reaffirming the principles of ethics involved.

The Council recommends that it, jointly with the Bureau of Medical Economics, be authorized to undertake these studies and to confer with other interested agencies, in order that it may be in a position to establish ethical standards for the practice of medicine by physicians holding positions in hospitals and to prevent the exploitation of either the public or the profession. If during this study it is revealed that hospitals registered and approved by the Council are exploiting the public or the profession, such approval may be revoked.

Respectfully submitted.

Ray Lyman Wilbur, Chairman.
John H. Musser.
Fred Moore.
Fred W. Rankin.
Charles Gordon Heyd.
W. D. Cutter, Secretary.

Report of Council on Medical Education and Hospitals

Dr. Ray Lyman Wilbur, Chairman, presented the following report:

There seems to be some confusion in the minds of a good many of the Delegates and members of the Association as to the exact function of the Council on Medical Education and Hospitals and various other organizations now in the field both of education and of the hospitals. This is important, since we are going into a period of considerable experimentation, legislative and otherwise. It seemed desirable for you to remember that the Council has its power through the Association and that that power is largely a matter of publicity. Our relationship to the institutions that teach medical students is primarily based on the fact that we publish an approved list of those schools. That gives us a position of influence if not of power. We cannot tell those institutions what to do. Our relationship to the hospitals is largely because we have to do with the education of interns and of resident physicians and publish a list of approved hospitals for interns and residents, and also a list of hospitals on a register. Through *The Journal*, and particularly through the Directory which we publish, we have a direct relationship to practically all the physicians in the country.

There has come into the field in recent years the advisory board of the various specialties. That advisory board has entered on a study of graduate instruction through a commission supported by the various foundations. The chairman of that commission is Dr. W. C. Rappleye. There are two members of the Council on Medical Education and Hospitals on that commission. It is to study the philosophy of graduate instruction, not those institutions giving such instruction.

There is also in this field the American Hospital Association and the American College of Surgeons, and each hospital is apt to have a relation to these three bodies.

In connection with the advisory board on specialties we have assumed certain powers in regard to them by not joining this advisory board but asking that each board submit to the Council its constitution and its program for the approval of the Council. Some of those who were reluctant to do so found that our power largely consisted in our ability not to list them in the directory, so that I think we can say now that all of the special boards are in a direct relationship to the Council.

The hospital is becoming the center of experimentation in the fields of medical care. It is said that there are some 300 different propositions in different parts of the country, and what goes on in the hospital is of enormous significance to us now as we approach this period when there is apt to be more and more in the way of legislative experimentation in this field.

There may be confusion between our Council and the American Hospital Association and the American College of Surgeons. We would like to have your continued support and an understanding with you that we as the representatives of the House of Delegates and the American Medical Association are in a position to take firm positions regarding certain of these propositions as they arise, and to seek the help of the other two associations. If you belong to the American College of Surgeons we ask you to help us bring about cooperation and coordination. If you are in the American Hospital Association we ask the same thing. If the medical profession in its relationship to these institutions is not a unit and not united, we are going to find ourselves in many difficulties that we can avoid by a well understood and well supported approach to these difficult questions.

I have made this statement in the hope that it would clarify for you just what is going on and so that it would leave in your minds the necessity of helping us represent you as the

special problems come up, as they will, in connection not only with hospitals but with the medical schools. This is of vital importance in the field of graduate instruction, since, as I suggested the other day, we are now getting support from various sources, including the federal government, in some of these graduate courses, and what goes on there will have to go on largely through the hospitals and our relationship with those hospitals must be not only understood but backed if we are to take this common program forward. Thank you very much.

Dr. Arthur J. Bedell, Section on Ophthalmology, moved that the report of the Council on Medical Education and Hospitals be approved, and the motion was seconded by Dr. H. B. Everett, Tennessee, and carried.

E. H. SKINNER

THE ROENTGENOLOGIST, THE PATHOLOGIST AND THE ANESTHETIST UNDER HOSPITAL INSURANCE PLANS

One of the most significant actions taken by the House of Delegates in San Francisco concerned the relationship of three professional groups—roentgenologists, pathologists, anesthetists—to various hospital insurance plans. Several resolutions which were offered from various sections of the country were considered by the Council on Medical Education and Hospitals. The Council in its report said:

The proposers of these resolutions, the delegates from the Massachusetts Medical Society, members of the California Medical Association and others, met with the Council to express their views concerning the problems that concern the practice of medicine in hospitals by radiologists, pathologists and anesthetists. These problems have been rendered more acute by the rapid extension of systems of group hospital insurance within the last few years. The Council believes that these problems are of vital concern to the medical profession; that unwise decisions at this time may lead to consequences that would be disastrous to physicians and to the public alike, and that, therefore, a serious study should be made of existing relationships between hospitals and the physicians practicing therein, especially in the departments of anesthesia, radiology, pathology and physical therapy, with a view to standardizing the relationship of these services to the hospital and, where necessary, of reaffirming the principles of ethics involved.

The Council recommends that it, jointly with the Bureau of Medical Economics, be authorized to undertake these studies and to confer with other interested agencies, in order that it may be in a position to establish ethical standards for the practice of medicine by physicians holding positions in hospitals and to prevent the exploitation of either the public or the profession. If during this study it is revealed that hospitals registered and approved by the Council are exploiting the public or the profession, such approval may be revoked.

This report of the Council on Medical Education and Hospitals was adopted by the House of Delegates of the American Medical Association. Therefore, during the coming year a suitable study will be made and recommendations no doubt will be drawn up for submission to the House of Delegates at the next annual session.

Until new recommendations are made and until the House of Delegates has adopted them, the policy of the Association in relationship to these matters remains unchanged. Should there be doubt as to the policy of the American Medical Association in this connection it is well to remember the action taken by the House of Delegates at San Francisco, which reads:

Since . . . some state and county medical societies may find it necessary to develop preferable procedures for supplying the needs where medical services are insufficient or unavailable, it is urged that these medical societies be guided in the development of these procedures by the ten principles adopted by the House of Delegates in 1934. The application of these ten principles to specific suggestions or proposals for the organization of medical services may be facilitated by utilizing the method of direct cash payments to individual members. Your committee unanimously concurs in the suggestion and recommends that the American Medical Association adopt the principle that in any place or arrangement for the provision of medical services the benefits shall be paid in cash directly to the individual member. Thus, the direct control of medical services may be avoided. Cash benefits only will not disturb or alter the relations of patients, physicians and hospitals.

Your committee has considered in detail that portion of the report of the Board of Trustees devoted to the Bureau of Medical Economics under the heading "Group Hospitalization" and also the separate statement of the Bureau of Medical Economics entitled "Group Hospitalization Insurance." Your committee commends the clarity and forcefulness of these statements and recommends that the ten principles adopted in 1934 as the policy of the American Medical Association be amplified by the addition of the following statement to Principle 4:

If for any reason it is found desirable or necessary to include special medical services such as anesthesia, radiology, pathology or medical services provided by outpatient departments, these services may be in-

cluded only on the condition that specified cash payments be made by the hospitalization organization directly to the subscribers for the cost of the services.

Disapproval of the inclusion of special medical services on a service basis in hospitalization insurance contracts will then be explicit but a constructive alternative arrangement will be possible.

These actions should make clear the point of view of the American Medical Association in this regard.

Recent apparently inspired news items have asserted that "the American Medical Association and various groups within the profession oppose the hospital insurance system which protects 1,600,000 Americans," The American Medical Association has never opposed hospital insurance; several of its constituent medical societies have been responsible for the development of hospital insurance plans. The plans prevailing in Washington, D. C., St. Louis, Oakland, Calif., and the statewide plans in Alabama and North Carolina were inaugurated by medical societies. In many other localities, medical societies are participating in the operation of hospital insurance plans.

The medical profession is certainly opposed to those undesirable types of hospital insurance plans which violate state insurance laws or state laws governing medical practice, and to those plans promoted for individual gain. Most of the plans which violated state insurance laws or which were operated for personal gain have been discontinued or reorganized. However, many hospital service plans continue to ignore state medical practice acts which require persons to possess certain educational requirements and pass an examination before they can offer to diagnose illnesses or treat the sick. Hospitals and hospital insurance corporations cannot fulfil these requirements. In most states the courts have therefore recognized that it is contrary to good public policy for a corporation to attempt to practice medicine by hiring physicians to practice medicine for the corporation. Physicians have always objected to those arrangements whereby a corporation (and most hospitals are corporations) attempts to sell the services of roentgenologists, pathologists, and anesthetists in a manner which results in a profit for the corporation or places the directors of the corporation in control of the personal relations between the patient and his physician. Similar undesirable relations are created when a corporation attempts to practice any profession. Consequently, the courts have also recognized that a corporation cannot practice law or hire lawyers to practice law for it. The same holds true for dentistry. These principles are maintained to give the individual person reasonable assurance that he will receive competent services from p ysicians, lawyers and dentists.

The action taken by the House of Delegates in San Francisco holds that it is desirable to permit each person to receive the benefits from a hospital insurance system in cash so that he may purchase his own medical services from whatever source he may desire. When the individual himself pays for the services he receives he can be assured of adequate and satisfactory services; otherwise he can refuse to make the payment. Under the cash-to-the-insured payment arrangement, which is the long-established method used by accident and health insurance companies, the insured person receives a definite number of dollars with which he can purchase services that are satisfactory to him. He does not have to accept whatever services may be offered by the insuring organization. The rights of the patient certainly are superior to those of the hospital, the corporation or any other interest.—*J. Am. M. Ass.*, July 9, 1938, *111*,.158.

AMERICAN CONGRESS OF PHYSICAL THERAPY

The seventeenth annual scientific and clinical session of the American Congress of Physical Therapy will be held cooperatively with the twenty-second annual convention of the American Occupational Therapy Association, September 12, 13, 14, and 15, 1938, at the Palmer House, Chicago. Preceding these sessions, the Congress will conduct an intensive instruction seminar in physical therapy for physicians and technicians—September 7, 8, 9, and 10.

The convention proper will have numerous special program features, a variety of papers and addresses, clinical conferences, round table talks, and extensive scientific and technical exhibits.

The instruction seminar should prove of unusual interest to everyone interested in the fundamentals and in the newer advances in physical therapy. The faculty

will be comprised of experienced teachers and clinicians; every subject in the physical therapy field will be covered. Information concerning the convention and the instruction seminar may be obtained by addressing: The American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago.

AMERICAN RADIUM SOCIETY

The Twenty-third Annual Meeting of the Amercan Radium Society was held in San Francisco at the Clift Hotel on June 13 and 14, 1938. The program was an interesting one and the scientific sessions were well attended. The banquet was held on Monday evening at which the Janeway Lecture was given by Dr. Henry Schmitz of Chicago on "Historical Retrospect of the Treatment of Carcinoma of the Uterus." At the conclusion of the lecture Dr. Schmitz was presented with the Janeway Medal.

The following officers were elected for the coming year: President: Dr. William P. Healy, New York City; President-Elect: Dr. Lawrence A. Pomeroy, Cleveland, Ohio; First Vice-President: Dr. W. E. Costolow, Los Angeles, California; Second Vice-President: Dr. Hayes E. Martin, New York City; Secretary: Dr. Frederick W. O'Brien, Boston Massachusetts; Treasurer: Dr. Charles L. Martin, Dallas, Texas. The announcement of the Twenty-fourth Annual Meeting will be given at a later date.

CORRECTION

The paper by S. W. Donaldson and H. A. Towsley on "The Incidence of Enlarged Thymus Following the Prenatal Use of Iodized Salt," June, 1938, issue of the JOURNAL, contains the following statement in the first column, last paragraph, page 913: " . . . Perkins [41] reported an incidence of 5 per cent of thymus in 500 children from one to two years of age in 1926." This should read "from one to twelve years."

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN AND RADIUM THERAPY

Mayer, E. G. Ueber das Zusammenarbeiten von Operateur und Strahlentherapeuten bei Tumoren der oberen Luft- und Speisewege von röntgenologischen Gesichtspunkten. (The cooperation of surgeon and radiologist in the treatment of tumors of the upper respiratory and digestive tracts from the roentgenologic viewpoint.) *Strahlentherapie*, 1937, *58*, 636–640.

In regions such as the nasal accessory sinuses, it is imperative to have operative drainage before instituting radiotherapy for cancer. Great aid is afforded in laryngeal carcinoma by a tracheotomy and a gastrostomy, in attempting to irradiate carcinoma of the larynx and stomach respectively. Without these aids the outlook is not only poorer but the general condition of the patient is made worse so that adequate therapy cannot be instituted.—*W. R. Stecher.*

Schwarz, G. Entwicklung, Prinzipien und biologische Grundlagen der röntgentherapeutischen Bestrahlungsmethodik. (Evolution, principles and biologic groundwork of roentgen therapeutic methods.) *Strahlentherapie*, 1937, *58*, 499–520.

It is well nigh impossible to abstract this amazing compact wealth of material, which in a few pages conducts one through the entire gamut of radiotherapy. This excellent article is recommended in the original; since the work is so fundamental and the very essence only reviewed, it is a veritable commentary. A panoramic discussion of the modern trends of radiotherapeusis, virtues and defects thereof, is especially worth while. One is left with the indelible impression that radiology is not even in its infancy, but rather in its embryonic form. The fundamentals of radiotherapeusis are perhaps more clearly outlined than in the original text, and their evaluation is very worth while. —*W. R. Stecher.*

Sgalitzer, M., and Ungar, E. Versuch einer körperlichen Eignungsprüfung zur Beschäftigung mit Röntgenstrahlen. (An attempt to test individual bodily reaction in persons whose occupation subjects them to roentgen radiation.) *Strahlentherapie*, 1937, *58*, 701–709.

A simple count of total number of leukocytes before, and on the eleventh and fourteenth day after the test irradiation (in this time the leukocytic diminution has reached its lowest point), offers a means of estimating the radiosensitivity of the leukocytes of an individual. It was found that different individuals presented varying reactions to irradiation, and that 30 per cent of 50 test cases showed a definite, albeit transient, leukopenia following a test irradiation. This is usually accompanied by a relative lymphocytosis. Control studies after many months showed this leukocytic radiovulnerability to be always, and only, in the same individuals. It was also determined that the small cumulative dosage such as occurs in persons in occupations dealing with radioactivity, i.e. radiologists, physicists, etc., showed a parallel effect on the leukopoietic system. Therefore, the authors conclude that this test should be performed on all potential persons dealing with radioactivity, and if they are found to be definitely sensitive, to strongly urge a different occupation. In this manner, the higher incidence of blood dyscrasias and untoward effects noted in radiologists, technicians, etc., will be, in some measure, avoided.—*W. R. Stecher.*

Stephens, D. J. Chronic myelogenous leukemia; observations before and during remissions induced by solution of potassium arsenite and by roentgen therapy with particular reference to bone marrow. *Am. J. M. Sc.*, July, 1937, *194*, 25–34.

Roentgen therapy and potassium arsenite have both been used in the treatment of chronic myelogenous leukemia; either has been found to produce temporary clinical remissions, reduction in the total and immature white blood cells, improvement in the anemia and reduction in the elevated basal metabolic rate. A few studies have indicated that during remissions, induced by either of these treatments, there is an actual return to red marrow. The author has studied the peripheral blood, the bone marrow, nitrogen balance and oxygen con-

sumption in 2 patients before and during such remissions.

The first case was of a single Italian girl who had had a laparotomy at the age of twenty-three because of subacute salpingitis. A few months later she was again hospitalized because of arthritis, which was thought to be gonorrheal. Two years later she was admitted with secondary syphilis. During the course of treatment for this some immature granular cells were noted (though repeated blood studies were negative for such, previously). Since early leukemia was suspected studies were frequently made; the hemoglobin was 14.5 gm.; red blood cells, 5,010,000; during the following three or four months, the white blood cells varied from 19,000 to 40,000 with the characteristic increase in immature forms. A biopsy of the sternal bone marrow was obtained. The patient was closely observed for the following eighteen months, by which time the leukocyte count reached 151,000. A second bone marrow biopsy was obtained and the patient placed on subtoxic dosages of Fowler's solution. The blood studies showed a return to normal, though some circulating myelocytes remained. A third bone marrow biopsy was obtained—this one, barely more than a month after the second one but at the height of the hematologic remission. Blood chemistry studies were also made.

The other case, studied in the same way, had been under treatment for myelogenous leukemia for almost four years. He had been given repeated courses of Fowler's and small amounts of roentgen therapy ("total 2,300 r"). When last hospitalized his white blood cells numbered 255,000 per c. mm.; red blood cells, 3,960,000; hemoglobin, 10.8 gm. Embryonic forms were numerous: myelocytes, 43 per cent; metamyelocytes, 13 per cent; 2 nucleated red cells in counting 100 whites. He had had no treatment for three months. A bone biopsy was obtained, and 1,000 r applied over the chest and abdomen, in divided doses, during a period of seven days. A second bone biopsy was obtained barely more than one month after the first—"at the height of the hematologic remission."

The progress of these 2 cases was the same: clinical improvement; restoration of approximately normal cytology; return of oxygen consumption to normal; replacement of gray, hyperplastic, cellular leukemic bone marrow by relatively hypocellular, fibrotic marrow with a markedly decreased myeloid-erythroid ratio. Roentgen therapy was followed by a marked increase in the nitrogen excretion which was not noted with the Fowler's regimen though both regimens produced weight loss.—*A. A. de Lorimier.*

Gould, S. E., Kullman, H. J., and Shecket, H. A. Effect of lead therapy on blood cells of cancer patients. *Am. J. M. Sc.*, Sept., 1937, *194*, 304–310.

The investigations of Blair Bell indicated beneficial effects by lead compounds in the treatment of cancer, and Bargen has recently reported encouraging results of the same sort. The authors have therefore studied blood changes resulting from the use of colloidal lead in a group of 22 cases of advanced cancer.

Colloidal lead triphosphate was given intravenously in doses varying from 35 to 140 mg.; the injections being given 2 or 3 times weekly over a period of two to four weeks. A progressive anemia developed in 16 of the 22 cases; the hemoglobin and erythrocytes decreasing at least 20 per cent with a pronounced drop in the color index. Anisocytosis and poikilocytosis of varying degree were noted in practically all the slides. Nucleated red cells were prominent in 20 of the cases. In many, reticulocytes exceeded 5 per cent. Polychromasia and achromasia were noted in most. Over 50 per cent of the nucleated reds showed basophilic stippling and 17 of the cases showed more than 1 per cent of the red cells stippled. Cabot ring cells were found 10 times in 6 cases; Howell-Jolly bodies, twice. Fourteen of the cases showed leukocytosis with an absolute increase in the neutrophiles (which rose to as high as 98 per cent of the total); lymphocytes were both relatively and absolutely decreased. Eosinophiles and basophiles were normal. One case showed a monocytosis of 11 per cent. The neutrophiles showed an increase in basophilic granulations. Myelocytes were found in 9 of the cases.—*A. A. de Lorimier.*

Levy, Robert L., and Golden, Ross. Roentgen therapy of active rheumatic heart disease. *Am. J. M. Sc.*, Nov., 1937, *194*, 597–601.

The fact has been recognized that roentgen rays exert a favorable influence on various forms of low grade infection. Rheumatic fever is known to be a chronic infectious disease which has a tendency for localization in the

heart. The authors have previously reported results of their treatment of this condition by roentgen irradiations. Improvement was indicated on the basis of electrocardiographic changes, relief of pains and general well being. The present report is being made on the basis of observations on 48 patients so treated, 12 of whom died. Fifteen of these patients were treated more than four years previously; several, as long as 11½ years previously. Twenty-five showed improvement; 3 were unimproved; in 8 cases, although recovery from the immediate attack ensued, there was no reason to believe that irradiation played a rôle. As for technique, 200 kv. (peak) was used, with a filtration of 0.5 mm. Cu plus 1 mm. Al; the field being large enough to include the heart; approximately 60 r (measured in air) was applied through the front of the chest and 100 to 125 r, through the back (depending upon the thickness of the individual)—the attempt being to distribute approximately 1/10 of the old "erythema dose" throughout the heart area. These treatments were given at intervals of two weeks, for 4 sittings. After a period of one to three months, this series was repeated. In general, those receiving the larger number of treatments fared the best. Cases which died showed numerous scars in the heart muscle and occasional Aschoff bodies but "no changes in the heart which could be ascribed to radiotherapy."

In general, it seemed that those patients with low grade activity responded better than did those with the more acute. Those with congestive heart failure appeared to be poor subjects for this type of treatment. Those with aortic insufficiency obtained no relief from pain.—*A. A. de Lorimier.*

Elsom, K. O'Shea, Miller, S. G., Forrester, J. S., and Chamberlin, G. W. Radiation and cholecystectomy as therapeutic procedures for typhoid carriers. *Am. J. M. Sc.*, Oct., 1937, *194*, 466–470.

Cholecystectomy has been found to render the feces negative to culture of the typhoid bacillus in chronic carriers who had previously shown positive stool cultures. Gulbrandson reported effective use of radiotherapy as a substitute for surgery; he found 4 out of 12 so treated to be freed of the infection, while the other 8 showed diminution in the number of typhoid bacilli in the feces. The authors have therefore carried out further studies of these measures.

Twenty-two carriers were included; 16 of these were women. The average age was 51.7 years; the youngest was thirteen; the oldest, seventy-seven. Only 13 gave a history of having had typhoid fever; an average of fourteen years elapsed between the illness and discovery of the carrier state. Except for one of these, who was a urinary carrier, the duodenal contents showed typhoid organisms, before treatment, and gallbladder function tests were impaired. Two submitted to surgery. Positive cultures were obtained not only from the bile content of the gallbladder at the time of surgery but also from the walls. Both of these cases later showed negative stools and duodenal drainages. Irradiation, directed over the gallbladders of 12 other patients, and over the kidneys of 1 urinary carrier failed to render any of them negative. The dosage varied from 650 r to 800 r for a series; 6 of the patients received two such series.—*A. A. de Lorimier.*

Sgalitzer, M. Welche der verschiedenen Methoden der Röntgenbestrahlung eignet sich am besten für die Behandlung des Zentralnervensystems? (Which of the various methods of irradiation is the best in the treatment of the central nervous system?) *Strahlentherapie*, 1937, *58*, 571–572.

In short, simple fractional treatments have been found the safest, for there is the danger of irreparable late effects in protracted irradiation. Generally in localized brain tumors, four large fields are employed. But one factor of great import is whether the irradiation is through an open or closed calvarium. A restricting factor is the elevation of intracranial tension, but in hypophyseal tumors and certain suprasellar tumors roentgenographic visualization can often best decide this point. All cases receive a test dosage of 50 r. The reaction of the patient is noted, and further irradiation planned accordingly. Never is more than one field irradiated per day. The highest dose never exceeds three times the test dose. The optimum effect in all brain tumors, except hypophyseal tumors is to be expected from surgical procedures rather than irradiation. If these principles are strictly adhered to, the author thinks that no untoward effects from irradiation will occur.—*W. R. Stecher.*

Schinz, H. R. Die fraktionierte und protrahiert-fraktionierte Bestrahlung. (Fractional and protracted fractional irradiation.) *Strahlentherapie*, 1937, *58*, 541–542.

The four methods are briefly described: (1) Simple fractionated short irradiation, in which the only effect is palliation. This is employed in advanced hopeless cases. The r/min. output is large, and the entire duration of treatment averages one week. (2) Protracted fractional short irradiation varies only in that the intensity is less, so that each single treatment is longer. (3) The simple fractionated long irradiation method consists of an intensity of 14–40 r per minute, and the duration of a series is over many weeks. (4) The method of choice in Zürich is the protracted fractionated long irradiation technique. The roentgen output is 2.5 r per minute; each treatment requires 60–80 min. The results with this method have been exceedingly gratifying in carcinoma of the air and food passages. Contrary to the majority of otorhinologists, the author holds this the method of choice in the treatment of carcinoma of the vocal cords.

The author employs adjuvant radium implantation in lip carcinomata, and particularly in carcinoma of the tongue. The intracavitary radium therapy is still the main method in carcinoma of the cervix uteri, but this is always preceded by roentgen irradiation of the parametria.—*W. R. Stecher.*

Schaefer, Walter. Die Methode der Kontaktbestrahlung in der Gynäkologie. (The method of contact therapy in gynecology.) *Strahlentherapie*, 1937, *58*, 606–608.

Schaefer employed contact therapy in gynecologic work in treating carcinoma of the cervix. His results, although compiled over a short period of time, are very encouraging. His experience over a period of three years, including cases treated in the last six months, showed statistics of 88 patients, with 47 per cent living and well. This compilation included all groups of involvement. The author concludes that the results as contrasted with radium therapy are similar if the absorption and the dosage remain approximately the same, and if future studies confirm this belief, this method will be a great advantage due to the expense and limited supply of radium. An added advantage is the irradiation during a laparotomy, which is not feasible with radium on account of its time factor. It is possible to angle the roentgen tube intravaginally to direct the rays "around the corner," as it were, into the parametrium. —*W. R. Stecher.*

Weibel, W. Ergebnisse der Kontaktbestrahlung nach Schaefer-Witte bei Karzinom der Frauenorgane. (Results with contact therapy by the method of Schaefer-Witte in carcinoma of the female genitalia.) *Strahlentherapie*, 1937, *58*, 609–610.

Weibel amplifies the method of contact therapy as noted in his practice. The group of 56 patients, (38 cases of which were inoperable), one year after contact therapy for carcinoma of the genital organs, showed fairly good results. The technique is to administer directly against the tumor 2,000 r in 10 sittings of 200 r. The parametrium is treated similarly over a period totalling six weeks and 30 sittings. Additionally external deep therapy is given, to attain a total of 1,500–1,800 r in the tumor, through two portals in 10 sittings employing a half-layer value of 2 mm. Cu and intensity of 7 r per minute. The total dosage in the tumor region approximates 3,500–3,800 r (6 H.E.D.), which is administered within four to six weeks. The intracavitary irradiation averages eighteen minutes per sitting. Although an intense local reaction of edema and reddening resulted, no untoward effects were noted.—*W. R. Stecher.*

Chaoul, H. Die Röntgennahbestrahlung (Niedervolttherapie), ihre Methodik und ihre Ergebnisse bei der Krebsbehandlung. (Contact roentgen therapy (low voltage therapy), methods and results in the treatment of cancer.) *Strahlentherapie*, 1937, *58*, 611–613.

Chaoul, whose method and modifications are in general use, reports his results in the treatment of cutaneous cancer in which in 135 cases 93 per cent were entirely symptom-free ranging from one to five years. In carcinoma of the lip in 37 cases his results showed 87 per cent symptom-free for a similar period. Malignant melanoma, strangely, showed 14 out of 17 cases symptom-free from one to four years. In carcinoma involving the buccal cavity, in 38 cases 42 per cent presented one to five year cures. In carcinoma of the glandular organs, in 58 cases 55 per cent showed one to three year cures. Carcinoma of the rectum again showed a surprisingly high survival rate: in 9 cases 7 were symptom-free from one to two years. In sar-

coma, 46 per cent of 13 cases had a one to three year survival rate. In totaling, one observes 307 cases treated by this method, 233 symptom-free (76 per cent), for the following duration periods—over 5 years (3); 4 years (14); 3 years (28); 2 years (54); 1 year and less (134). In evaluating this method as compared to radium therapy, approximately similar results are obtainable in skin and lip carcinomas, in which the lesion is not infiltrating. In definitely infiltrating lesions contact roentgen therapy affords better results. In carcinoma of the buccal cavity approximately identical results are obtainable. Of greatest encouragement is the efficacy noted in roentgen therapy of malignant melanoma and inoperable rectal carcinoma, which heretofore were most refractory to radium therapy. The author foresees further use of this method in cancer of the gastrointestinal tract and other tumors within the body cavity, if they can be made accessible to direct irradiation.—*W. R. Stecher.*

Frank, A. Beitrag zur Nahbestrahlungsmethode. (Report on the method of contact irradiation.) *Strahlentherapie*, 1937, *58*, 618–622.

Frank presents excellent illustrations demonstrating the results he obtained in using contact therapy. He also has devised a minute ionization chamber which registers the roentgen output during treatment. He again stresses the fact that total doses of 10,000 r have been administered to the skin without latent untoward skin damage. The technique is essentially that of daily treatments of 200–500 r until a total dose of 6,000–8,000 r is administered. Hemangioma of the buccal mucosa and plantar warts showed excellent response to this therapy.—*W. R. Stecher.*

Melchart, F. Die weitere Entwicklung der Kontakttherapie. (Further experiences with contact therapy.) *Strahlentherapie*, 1937, *58*, 614–617.

Melchart believes that roentgen therapy will not entirely supersede or supplant radium therapy, but only on account of physical factors of unapproachability. Thus, the radium bomb is definitely being supplanted by teleroentgen therapy, and where contact therapy can be used in lieu of radium, equally as good results are procurable. The one advantage of radium is of course its interstitial use. The economic factor is of greatest importance in considering the advantages of contact therapy over radium, and particularly the time-intensity factor. Theoretically, the shorter wave lengths of radium and resultant Compton effect in the tissues should attest to its superiority over the relatively long wave lengths of the low voltage contact therapy method; however, clinically and experimentally this has not been found to be demonstrably effective. Thus one is led to believe that there is no particular virtue in the type of radiation *per se* over a certain beam of wave lengths as utilized practically to date. The author speculates that the supervoltages, i.e. 1,000 kv. roentgen therapy, only will be more useful as regards the amount of radiation administered rather than quality; thus a somewhat better depth dose is obtainable and only that. He also mentions that soon 2,000 kv. will be practicable due to the refinement in mechanical construction. Contrary to Chaoul's dictum, Melchart considers a shortening of the focus-skin distance from 5 cm. as an advantage in certain cases where a very superficial intensity is desired. Thus, where radium was 7 mm. from the skin there was 51 per cent in 0.5 cm., 29 per cent in 1.0 cm., and 13 per cent in 2.0 cm., as contrasted with a focus-skin distance of 2.0 cm. where 50 per cent at 0.5 cm., 26.4 per cent at 1.0 cm., and 11.1 per cent at 2.0 cm. was procured.

The author predicts great advances in mechanical control of this method, in the form of smaller tubes, enabling the operator to approach hitherto inaccessible cavities. Thus lowering the voltage will not greatly handicap the method, particularly in treating laryngeal carcinoma after a Coutard series has been completed, for the destruction of the residuum. At present there is experimental work with an air-cooled contact therapy apparatus and this will be one advance.—*W. R. Stecher.*

Merio, P. Bericht über die Erfolge der Kontakttherapie an der II. Chirurgischen Klinik. (Note on the results of contact therapy in the Second Surgical Clinic.) *Strahlentherapie*, 1937, *58*, 623.

Merio reports 12 neoplasms treated by contact therapy, utilizing doses of 6,000–12,800 r. Inasmuch as all were inoperable, the results, namely 5 greatly improved, were considered rather encouraging.—*W. R. Stecher.*

Gunsett, A. Methode der Bestrahlung der Krebse mit extrem hohen Spannungen. (Methods of supervoltage radiation therapy of carcinoma.) *Strahlentherapie*, 1937, *58*, 573–592.

An excellent description of this newer method of radiation therapy apparatus is given, together with exact data regarding the output of this 800 kv. generator, the effect of potential, size of field and filtration upon the depth dose, back-scatter and cutaneous reactions therefrom. In increasing the potential from 200 to 520 kv. (peak) the increased depth dose procurable was 30 per cent; the back-scatter definitely diminished and the cutaneous reactions correspondingly definitely decreased, which factors permitted of greater application of radiation to the neoplasm. This last factor is probably of more importance than hitherto thought, for the relative increased depth dose *per se*, in increasing the potential, could possibly be attained by other physical factors employing the more commonly utilized 200 kv. potential. The field of usefulness is obviously perhaps limited to deep-seated cancer. Of further importance in comparing 200 with 520 kv. radiation is the rather striking drop in leukocytes and increased sedimentation time when employing the higher potential. This of course was anticipated; but none the less must always be considered as a shortcoming. All in all, the author is highly optimistic as to the increased value of this supervoltage therapy and thinks that it will be soon in general use, for the cost of this type of equipment will soon be greatly lessened.

The type of malignant disease is of course an important consideration when deciding as to the relative practical merits of low and high voltage therapy. Thus, a radiosensitive tumor is perhaps better treated from theoretical grounds by high voltage therapy whereas a radioresistant tumor, under no conditions, can be made more sensitive, even though the increased dosage in the tumor procurable thereby may be sufficient to bring about recession, at least. The refractory malignancies such as gastric carcinoma, intestinal malignancy, prostate and bladder carcinoma, and bronchial carcinoma are fertile fields for trial of this type of treatment. One would think, upon superficial examination, that extremely hard radiation is comparable in usage to telecurietherapy; but this is not the case. Advantages of high potential roentgen irradiation are, first, the economic difficulty in obtaining sufficient radium, and secondly, in the author's 2 gm. radium cannon, with a focus-skin distance of 9 cm. only a 30 per cent depth dose (10 cm.) is obtained and with the 5 gm. cannon at 11.0 cm. focus-skin distance a depth dose of 33 per cent. This is useful only for not too deeply situated malignancies, such as lymph node metastases, pharyngeal, laryngeal and palatine carcinomata, and above all, mammary carcinoma.

Statistical comparisons are not procurable to date as insufficient cases have been treated and sufficient time has not elapsed.—*W. R. Stecher.*

Wintz, H. Die Einzeitbestrahlung. (Single dose irradiation.) *Strahlentherapie*, 1937, *58*, 521–527.

For twenty years the author has been a staunch advocate of the single massive dose method, the aim being to apply the entire radiation in as short a time as feasible. Thus, to date, one series of treatment for cancer consists of four to five hours' duration in carcinoma of the uterus, followed in six to eight weeks by similar irradiation directed to the parametria. In this manner, great economic saving is made possible, the patient being confined to the clinic generally five to six days. This method requires exactitude in dosage measurement and directing, as a slight error would be greatly magnified. The author is very dogmatic in his opinion, which is very convincing, that the principle of massive single dosage is the most propitious method in obtaining a carcinoma lethal dose. All forms of fractionated dosage are discarded, not only upon theoretical grounds, but by actual clinical evaluation. It is apparent that the total irradiation in the pelvis is relatively small compared to the Coutard protracted method; but this should not mislead one, because the time element is of great importance not especially in the radiovulnerable karyokinetic phase, but in the general neoplastic radiosensitivity compared to normal tissue, which is considered maximum on initial irradiation. As an illustration, it is shown that the skin similarly tolerates protracted irradiation much better, and this phenomenon it is inferred might well be true of the neoplasm. To date, this disparity has been explained on the basis that the skin will accumulate radiation and tolerate insult more than the malignant cells. However, in this massive dose method the skin is in good condi-

tion after treatment, with but slight pigmentation without thickening and induration. Further no irreparable alteration in the pelvic connective tissue, etc., is noted. Of course the economic consideration is to be thought of; but this is not of paramount importance. Statistics are appended, which are very inclusive, in that all cases are presented regardless of whether complete or incomplete therapy was given. All cases are proven carcinoma; and only five-year cures being considered as those who are symptom-free. One is surprised at the report when one considers the results reported, and the controversy over the form of therapy affording the best results becomes a question far from settled.

	Cures *Portio carcinoma* (5 yr. after massive dose roentgen therapy)
Operable cases—186	114—61.3%
Inoperable cases—1078	136—12.6%
Total	250—19.7%

	Cures *Adenocarcinoma Corpus Uteri* (5 yr. after massive dose therapy)
Operable cases—104	71—68.2%
Inoperable cases—93	12—12.9%
Total	83—42.1%

Statistics for mammary carcinoma are presented in tabulated detail, and briefly in the Steinthal I and II groups: 47.7 per cent 5-yr. cures were attained; and in Steinthal Group III, 14.1 per cent 5-yr. cures were shown. This article is well worth reading in the original, before fully accepting the newer forms of therapy.—*W. R. Stecher.*

Holfelder, H. Methodik der Sättigungstherapie, besonders der Frankfurter Modifikation mit fallenden Dosen.(The saturation method, especially the Frankfurt modification entailing diminishing doses.) *Strahlentherapie.* 1937, *58*, 528–536.

A rather short yet embracing discussion of the currently employed methods of irradiation, namely, "Pfahler-Kingery saturation technique," and "Coutard's protracted fractional method" are discussed, as well as the author's modification which is somewhat comparable to a happy combination of both. Fundamentally, the present concept of irradiation emphasizes the tumor base which must be kept intact and not damaged by excessive irradiation. In this sense, a cancericidal dose *per se* probably does not exist, even though a lethal dose averages theoretically 5,000–8,000 r. It is always probable that a few cells are more radioresistant than the mean determined, and will resist very large amounts of radiation which would cause untoward damage to the surrounding tumor bed, which should be preserved to kill these remaining cells. It is of paramount importance, and unfortunately not sufficiently appreciated in general, that the closer the tumor is brought to the skin, the greater the effective dosage that can be applied. Actually this can only be accomplished conversely, i.e. by compression. This factor plus the preservation of the tumor bed by tangential irradiation in breast carcinoma, etc. is of great importance. It becomes difficult for the average patient to tolerate undue pressure when the so-called Coutard technique is employed, due to the protraction of the treatment. Experimentally, the same has been accomplished by further fractionating the treatment, and experimental work proving this is submitted. Thus, fractionation permits of more than 10 times an erythema value to be applied to the skin without damage over a period of four to eight weeks. Of more importance is the intensity (r per minute). The smaller the fractional doses become, the greater is the tolerance of the tissue.

The Frankfurt method, in substance, is as follows: compression to maximum effect is employed, with relatively short periods of exposure. Six to eight fields are utilized, each no larger than tumor bed. Daily dose per field is 300 r; the first three days two fields are irradiated daily; and then from fourth day on, each day one field is treated. After three weeks only every second day is a field treated. In this manner an initial saturation dose is arrived at with gradual reduction in dosage. This technique is conducive to the best results, in the author's experience.—*W. R. Stecher.*

Weber, E. Über die einfach-fraktionierte Bestrahlungsmethode. (Simple fractional radiation therapy.) *Strahlentherapie*, 1937, *58*, 557–559.

It is an economic impossibility to conduct therapy along the lines outlined by Coutard,

namely, two-hour treatments, in most clinics. The author's technique consists of 180 kv., 4 ma., 0.5 mm. Cu plus 1.0 mm. Al, 30–35 cm. skin-target distance, 25 r per minute. The fields are made as small as possible. In undifferentiated and non-infiltrating carcinoma the irradiation time is twenty days, in contrast to the differentiated and infiltrating forms requiring forty-five days with diminished daily dosage. If the author includes the early, small isolated non-infiltrating malignancies of squamous cell type involving the epiglottis and vocal cords, without lymph node metastasis, then the complete disappearance of the tumor would approximate 100 per cent. In pharyngeal and laryngeal tumors with infiltration and lymph node involvement, by means of increasing the time factor to forty-five days, 45 per cent of the cases showed complete disappearance of the neoplasm. When extensive spread had occurred the results showed only 5 per cent cured. All statistics obviously depend mainly upon how early the lesion is treated rather than upon the method. No marked late reactions were noted in the series treated. Coutard is perhaps correct in his emphasis upon the factor, of the importance of dosage *per se*, in undifferentiated and non-infiltrating cancers. In the opposite type, i.e. differentiated, infiltrating carcinoma, the time factor becomes more important than the total dosage in itself, and small, long-continued doses are applied. The production of radioepithelitis is a positive sign that the irradiation has been sufficient in undifferentiated and non-infiltrating carcinoma. It must be remembered that the therapeutic and toxic doses even with fractional irradiation, are rather close together, and when epithelitis occurs, the author increases the length of time and amount of final treatments.

Local recurrences after radium therapy of carcinoma of the tongue show good results with further fractional roentgen irradiation. Overdosage, with local tongue and osseous necrosis, must be constantly thought of and averted. In inoperable carcinoma of the thyroid, only palliation was obtainable with the protracted treatment.

Circumscribed bronchial carcinoma showed some response to this type of therapy, but the metastasis was not prevented. In inoperable mammary carcinoma, with shortened time factor, the results were very favorable. When malignant cachexia has presented itself, particularly in parametrial extension with anemia, the patient does not tolerate treatment well. Similar experience was had after blood transfusion. The poor results are chiefly attributable to the type malignancy *per se*, rather than amount of tissue irradiated, blood damage, visceral damage, etc.—*W. R. Stecher.*

Berkman, T., and Dessauer, F. Versuch, die fraktionierte Bestrahlung durch Provokation sensibler Zellphasen zu unterstützen. (An attempt to accentuate the sensitive cell phase during fractional irradiation.) *Strahlentherapie*, 1937, *58*, 551–556.

Provocative irradiation is the term for this procedure. The work is not new but merely an attempt at modification of other tried methods. If one accepts the principles of Regaud and Coutard, then the results can be bettered by (1) application of greater dosage to focus involved by protraction and fractionating; (2) if the incidence of irradiating the cells in their karyokinetic radiosensitive stage is increased. A review of the numerous attempts at increasing the radiovulnerability by various agents such as ultraviolet light, mustard oil, diathermy and galvanization, active hyperemia and cholin injection is given. Combining radiotherapy with diathermy showed no remarkable increase in betterment of results. Short wave therapy has likewise been employed jointly with irradiation.

It is common knowledge that the primary and secondary effect of irradiation varies with tissue involved, and the interval between depends considerably upon the dosage, thus 170 r (72 hr.), 300 r (192 hr.). Actual acceleration of cell mitosis can be accomplished by heat, and a survey of cellular growth during winter and summer months is Nature's example to follow. After primary irradiation, the mitotic count recedes; after an interim this rises to a maximum. The exact time for this to occur for various tissues in not definitely known. But small doses, as outlined by the Coutard method, show an interval of one or more days, until ascent of mitotic percentage occurs. It is during this time that diathermic heat is appied to accelerate this factor (½–1 hr.). After an interval of waiting, further irradiation is applied. Briefly the physical factors employed were as follows: 200 kv. constant potential; 0.8 mm. Cu and 1.0 mm. Al; 30–40 skin-target distance. Only refractory cases were studied with this method

Twelve cases are briefly discussed from which, of course, no definite results can be deduced.—*W. R. Stecher.*

Wieser, W. Zur Methodik der Schwachbestrahlung. (The method of irradiation with weak doses.) *Strahlentherapie*, 1937, *58*, 646–650.

The principles of treatment of inflammatory diseases by radiation are very briefly considered. A generalization is made by the author as to the dosage in acute and chronic cases. In the former, the dosage varies from 15–100 r in the diseased site; whereas in the chronic inflammatory processes, 1 to 100 r is given per sitting. There is an omnipresent danger of cumulative effect in every case of prolonged treatment. In treating chronic inflammations, one should not be guided by the immediate response of the lesion as in the acute inflammations; for it usually requires days and weeks for visible effects. Therefore, intervals of treatment should always be prolonged in cases where a long series is anticipated to prevent overdosage. Numerous examples are given to illustrate the necessity of individualizing each case. In one case of multiple sclerosis, a rather large single dose was given with favorable reaction within a very short period; however, within a year the signs and symptoms all returned, and the lesions were radioresistant. If small doses are given this recurrence does not take place. The same is true for epilepsy and other forms of chronic inflammatory affections.—*W. R. Stecher.*

Quastler, H. Die Wirkung herdferner Bestrahlungen in der Röntgentherapie tuberkulöser Lymphome. (The effect of distant roentgen therapy on tuberculous lymphoma.) *Strahlentherapie*, 1937, *58*, 688–693.

This is not a new discovery, but a worth while amplification of roentgen effects that to date have had no accepted explanation or absolute proof. Thus, in treating the spinal root areas in certain skin diseases, the question as to its modus operandi has repeatedly been questioned, one school maintaining that the effect is merely skin and tissue reaction acting systematically, rather than any action on the nervous mechanism *per se*.

The exposure of distant skin tissue, the author terms "adjunct" therapy. Thus, two (20×20 cm.) fields, anterior and posterior, upon the abdomen and back respectively, are irradiated twice on four successive days, with factors of 70 kv., 2.5 mm. Cu filter; 40 cm. skin-target distance, 200 r per field, with an intensity of 10 r per minute. The endeavor is obviously the application of weak radiation for skin and subcutaneous absorption. Two to four days later, the tuberculous lymphoma is treated directly by a rather similar technique with less initial dosage, amounting to 75–100 r in the lesion. The author's experience attests undeniably to the efficacy of this combined therapy, although no tenable explanation is forthcoming to date. However, experiments disclosed that adjunct therapy alone afforded no regression in the primary tuberculous lesion. The rationale is considered by the author as an increased radiovulnerability by this mechanism.—*W. R. Stecher.*

Sluys, F. Die totale Röntgenfernbestrahlung. (Total teleroentgen therapy.) *Strahlentherapie*, 1937, *58*, 599–602.

The optimum distance for this type of work is considered 2–2.5 meters as 1–1.5 meters cannot be considered true teleroentgen therapy, and farther distances than 3.5–5 meters show no effective difference, with much added expense of operation. The most practical filter was found to be 0.5 mm. Cu plus 2.0 mm. Al. As a generalization, doses should never exceed 50 r, and in lymphogranulomatosis malignum, even 15 r is often attended with reaction and poor results. It is important to note that in the leukemias, the blood picture and symptomatology may be very divergent; and the blood picture *per se* must not be treated. At times, there is a sudden drop in leukocytes without symptomatic relief, and further irradiation is fraught with danger. Possibly the best guide is symptomatology, carefully guided by any alarming blood picture change as noted by a hematologist. In a normal individual this type of irradiation manifests itself initially in a tendency to disappearance of lymphocytes, increased eosinophiles, relative increase in polymorphonuclears, and marked increase in blood platelets. In most cases a temporary increase in red blood cells occurs, but with increased irradiation a marked and serious anemia ensues.

In myeloid leykemia, startling results are effected by teleroentgen therapy. The recession in hyperleukocytosis is as rapid as in the older methods, and there are the added advantages of quicker response of red blood cell increase, and more rapid return to normal leukocytic

morphology. The length of life is definitely increased with this form of therapy. Also, the frequent detrimental radioresistance, which accompanied the older forms of treating individual bones and spleen, is less prone to occur.

Lymphatic leukemia is perhaps the ideal form of dyscrasia for this type of therapy. Caution must be exercised during treatment for any suggestion of a rapid erythrocyte fall; and small transfusions are in order to combat this.

Polycythemia rubra vera (Osler-Vaquez' disease) shows unpredictable and irregular response to irradiation. Here the leukocyte count must be carefully watched for any precipitant fall, for even without treatment it is not unusual to have a concomitant hyperleukocytosis rapidly disappear. If other clinical methods have been ineffective, further roentgen therapy has not been conducive to much success, possibly due to acquired radioresistance.

Localized Hodgkin's disease is rare; the common form is generalized. The bizarre course of this disease suggests a protean etiology, viz., tuberculoid infection, or chronic infectious process; yet at other times very definitely that of a tumor, i.e. lymphoblastoma. Certain cases have been reported living and well after ten years, one cited by the author twelve years with two viable children born in the interim. The common pseudo-infectious form, with signs and symptoms of a febrile infection, namely, Pel-Ebstein fever, weakness, diffuse aching and pruritus, and gastrointestinal disorders must be carefully watched when treated radiotherapeutically, particularly if large fields are employed, inasmuch as this type of patient is very ill. In those cases with paucity of symptoms, and polymicroadenitis, teleroentgen therapy affords excellent results, and particularly if tissue is irradiated in which no visible involvement is present. Here above all, one must remember the maxim of as "little radiation as is necessary." It is imperative to have what appears to be synergistic arsenic therapy concomitantly with irradiation.

Regarding highly malignant tumors, such as seminoma, lymphocytoma, sarcoma, this form of therapy suggests some improvement over the former classical treatment. In advanced carcinoma with generalized metastasis, especially to the skeletal system this distant irradiation is very successful from a palliative standpoint. Nothing definite to date can be stated in regard to teleroentgen therapy in endocrine disturbances, but this phase of therapy is being given careful thought and experimental control.—*W. R. Stecher.*

Schreiner, Bernard F., Reinhard, Melvin C., and Wehr, William H. Tele-curie-therapy. *Am. J. Cancer*, June, 1935, *24*, 386–392.

The authors describe the methods of treatment of surface and deep lesions by means of single or multiple radium packs at a distance. The treatments are fractionated and the dosages recorded in roentgens. Isodose charts for a single section and a triple section pack are presented. The authors also present a table giving the percentage intensities down to a depth of 20 cm. delivered by both single and triple section packs at 10 cm. radium skin-distance and also by a single 4 gm. radium pack at 6, 10, and 15 cm. radium skin-distance.—*J. S. Weber.*

Freund, F. F. Zur Klinik und Therapie beruflicher Röntgenschäden. (Clinic and therapy of professional injuries resulting from roentgen rays.) *Strahlentherapie*, 1937, *58*, 694–696.

In brief, all those persons engaged in radiology and subject to irradiation should have frequent check-ups on their blood count, for the first suggestive sign of chronic irradiation effect is a drop in the leukocyte count with relative lymphocytosis. When recognized at this stage, the prognosis is excellent; for further protection from the radiation, a period of vacation and general rebuilding is all that is necessary. The author recommends physical exercise and vacation at higher altitudes if feasible. As to drugs, the author considers arsenic and various forms of amidopyrine as contraindicated and to be especially avoided when prescribed for coincidental ill health.—*W. R. Stecher.*

Martin, Hayes E., and Stewart, Fred W. Spindle-cell epidermoid carcinoma. *Am. J. Cancer*, June, 1935, *24*, 273–298.

Spindle-cell epidermoid carcinoma is frequently difficult to diagnose. Its occurrence has been observed in animals as well as humans. The spindle-cell metaplasia appears to be a type of scar tissue carcinoma and most frequently occurs in areas which are the seat of pronounced changes as a result of prolonged roentgen irradiation. In the 8 cases presented,

4 had had numerous roentgen treatments to the skin of the face over long periods of time, 2 had had roentgen treatments within the last year or so, and 1 had had radium treatments about three years before. Cautery or endothermy, especially at the site of previous roentgen change, seems to cause the production of these cancers, for they will arise in the exact location. In 1 case, there was a history of a contused wound with possibility of foreign body.

Spindle-cell epidermoid carcinomas usually grow slowly, although their course may be very rapid. The margins are indistinct and therefore excision must be done widely. Since cautery is a predisposing cause, surgical excision is best. Irradiation is contraindicated since it likewise is a predisposing cause and the lesions have been found to be radioresistant. Neck dissections should be performed if there is palpable metastasis, although in this series of cases, after metastasis to the lymph nodes had occurred, the prognosis became hopeless.

The 8 cases are reported in detail and the histogenesis, etiology, clinical features, treatment and their results are fully discussed. Eighteen plates and a good bibliography are included.—*J. S. Weber.*

GOEDEL, R. Die Röntgenbestrahlung der puerperalen Mastitis. (Roentgen therapy of puerperal mastitis.) *Strahlentherapie*, 1937, *58*, 651–655.

Roentgen therapy is the method of choice in acute mastitis. With his technique the author had 29 rapid cures in 30 cases, a percentage of 97. In the remaining case, incision was necessary. In contrast, heretofore in this clinic, 90 per cent of the cases were treated surgically. The technique consisted of small doses (50 r), administered every third or fourth day, for a total of 3–4 treatments. In certain cases a single dose of 75 to 100 r was used, but the author strongly recommends the smaller, divided dosage.—*W. R. Stecher.*

COOPER, ZOLA K., and SEELIG, M. G. The effect of radiation upon reticulum in squamous cell carcinoma of the uterine cervix. *Am. J. Cancer*, May, 1937, *30*, 32–38.

Thirty cases of squamous cell carcinoma of the cervix were studied in the following manner. A biopsy was done, followed by roentgen therapy delivered at 200 kv. to tumor doses ranging from 418 to 2,910 r. One month later a second biopsy was taken, followed immediately by radium application totaling from 2,768 to 6,000 mg-hr. Eight weeks later the third and last biopsy was taken.

The authors conclude that: no correlation is demonstrable between the amount of reticulum and the radiosensitivity of the tumor or the clinical results following roentgen treatment; that there is some indication that an inconsequential increase in the amount of reticulum occurs following the application of radium but neither roentgen rays nor radium has any qualitative histological effect upon reticular fibrils.—*J. S. Weber.*

SCHREINER, BERNARD F. Successful irradiation treatment of eight cases of inoperable rectal carcinoma. *Am. J. Cancer*, June, 1935, *24*, 326–333.

The complete case report together with photomicrographs of the biopsies of each case is reported. The method of treatment varied widely. Both radium and radon were used within the rectal lumen. External irradiation was administered by means of "low power" roentgen rays, "high power" roentgen rays (200 kv.) and telecurietherapy. Interstitial irradiation was administered by means of radon in both glass seeds and gold seeds.

Two or more of the above methods were utilized in each case. Four of the cases are living and well from nine to 13½ years; two of these have strictures which, however, do not interfere with normal bowel action. One other case is living and well five years and four months. One case died of pneumonia at eleven years and nine months with no evidence of disease. This was the only case in which colostomy was necessary for rectal stricture. One case died at 7½ years of liver metastasis which had been present for two years but with no evidence of local disease at autopsy.—*J. S. Weber.*

MISCELLANEOUS

FURTH, J., and FURTH, O. B. Neoplastic diseases produced in mice by general irradiation with x-rays. 1. Incidence and types of neoplasms. *Am. J. Cancer*, Sept., 1936, *28*, 54–65.

The relative frequency of leukemia among radiologists suggested to the authors that the incidence of leukemia in their mice colony might be increased by irradiation with roentgen rays.

For this experiment, three stocks of mice were used; 775 out of 2,068 were irradiated. Each mouse received a single dose or several doses of from 200–400 r, the factors being, 190 kv., 30 ma., 50 cm. distance, and 0.5 mm. copper and 1 mm. aluminum filtration. The time intervals between the irradiations were from three to five weeks. The neoplasms developed five months or longer after irradiation. These occurred mainly in the organs severely injured by the roentgen rays. The irradiation caused an increase in ovarian tumors to fifteen times as many as in the controls; of myelosis, to eight times; of mediastinal lymphosarcoma to seven times.

Breast tumors, on the other hand, were diminished by one-half. This reduction was attributed to the decrease in the ovarian hormonal activity. The high incidence of breast cancers with ovarian neoplasms and the associated mitral hyperplasia suggested that some ovarian tumors produce hormones. During the experiment it was noted that roentgen rays, in the amounts used, greatly increased the susceptibility of the mice to infection.

Roentgen-irradiation induced tumors are indistinguishable from those occurring spontaneously. It is suggested that roentgen rays so alter the constitution of certain cells that they or their offspring undergo malignant transformation several months after irradiation.—*E. E. Downs.*

Sugiura, Kanematsu. The effect of roentgen rays on the growth of mouse sarcoma 180 irradiated *in vivo*. *Radiology*, Feb., 1937, *28*, 162–171.

Experiments were conducted to show the effect of filtered radiation upon transplantable mouse sarcoma No. 180 *in vivo*. The authors previously showed that about 2,800 r of filtered radiation was a lethal dose for this sarcoma *in vitro*. As a preliminary study it was found that normal mice will withstand about 350 to 400 r in a single massive dose of radiation to the entire body. However, if only a small area (2.9 cm. sq. or 4.8 cc. body volume) is irradiated, dosages to 3,000 r may be given without harmful effect upon the general body condition.

The physical factors used were 200 kv. (peak), 30 ma., 0.5 mm. Cu plus 1.6 mm. Al, 50 cm. distance and the output was 59 r per minute. It was found that 1,480 r or over, regularly produced a definite inhibition in the growth of the sarcoma, with subsequent complete regression in about twenty-four days. The wide difference in radiosensitivity between this tumor *in vivo* and *in vitro* would indicate that the destructive effect of irradiation upon tumors *in vivo* is due to a combination of direct action on the tumor cells and the reaction of the surrounding tissue.

As a part of this study an investigation was made to determine the transplantability of mouse sarcoma No. 180 into irradiated areas in mice. A small part of the body of a mouse was irradiated with 1,500 r and at the end of definite intervals of time, untreated tumor fragments were inoculated into the irradiated area. It was found that a high percentage of tumor transplants failed to grow if the implantation was done within two hours after irradiation; however, if the implants were made seven days after irradiation, the number of takes was almost 100 per cent and the rate of tumor growth was almost normal.—*J. H. Harris.*

Forfota, Erich. Röntgentherapeutische Versuche an erythroleukotischen Hühnern. (Roentgen therapy in erythroleukosis in chickens.) *Strahlentherapie*, 1937, *58*, 295–305.

Chickens were given various quantities and qualities of radiation, the average dose being 15 per cent to ⅓ S.E.D. In no instance was there any definable effect on the course of the disease, length of life of the affected chicken, or diminution in virulence or infectiousness of the agent. Each generation was given roentgen radiation and there was no weakening of virulence up to the fifth generation. Relevant to the erythroleukotic hemogram, it was noted that small doses were less effective than larger doses of radiation, and that more highly filtered radiation was somewhat more effective; but in no case was there any sustained arrest of the overwhelming leukocytosis, or the increasing presence of pathological cells from the hematopoietic system in the blood stream. A comparison made between the radioresistance of erythroleukotic blood picture and true leukemia shows the marked resistance of the former compared to the latter.—*W. R. Stecher.*

Dahl, B. Tierexperimentelle Untersuchungen über die epilatorische Wirkung der Röntgenstrahlen in ihrer Beziehung zur Röntgendermatitis sowie über den Einfluss der Bestrahlungstechnik auf diese Wirkung. (Experimental determination of the epilation dose of roentgen rays in relation to roentgen

dermatitis, and the effect of various radiation techniques.) *Strahlentherapie*, 1937, *58*, 336–344.

In recent experimental work the epilation rather than the erythema dose has been employed in irradiation studies. The tolerance of animals to radiation has been found to be approximately two and a half times greater than in humans. Various reaction grades to irradiation have been formulated, but the author considers the stage of exudative dermatitis as the most accurate criterion for comparative radiosensitivity experiments. In humans, partial epilation occurs in from 25–40 per cent of this value; and complete epilation from 50–75 per cent. It is noteworthy that permanent alopecia only occurs if a very severe exudative dermatitis ensues.

Both protraction and fractionation of the irradiation diminished the effectiveness of the dosage, but no change in relation to appearance of epilation and dermatitis was noted. Various qualities of radiation were employed, and similar doses produced identical reactions provided the radiation intensity was constant. Holthusen's hypothesis that "the epilation effect of a dose of radiation at one sitting is the same for all intensities between 500 r/min. and 2 r/min., whereas, the erythema effect of dosage diminishes with weakened intensities" was borne out.—*W. R. Stecher.*

OTTO, ERNST. Anmerkungen zu Dosierungsfragen. (Concerning dosage questions.) *Strahlentherapie*, 1937, *58*, 361–367.

A very illuminating discussion of the many current conflicting views of biologic radiation reaction is presented. The writer observed as high as 300 per cent difference in the skin erythema dose value in various clinics; but the main factor he considers, the evaluation of the S.E.D. in regard to the intensity of the radiation, namely the time factor.

Formerly, the common opinion was that various wave lengths produced different biologic effects. Many authorities are quoted showing the general trend of adjudicating the greater tolerance of the skin to "hard" radiation, the S.E.D. for "soft" radiation being considered 450 r compared to 600 r for "hard" rays. Recently American observers have considered the skin reaction to filtered rays less than to unfiltered rays. However, opponents to this generalization are not lacking; for certain radiologists are strongly of the opinion that there is absolutely no difference in biologic action among the various wave lengths. Holthusen considers the amount of absorbed energy for the formation of erythema, regardless of wave length, and gives the S.E.D at 800 r with intensity between 15–50 r per minute.

Schwarz goes still farther in stating that not only are hard rays similar in biologic action, but definitely more harmful to the skin, due to the increased energy absorption. This might be explained upon the basis that the short wave lengths act relatively more on the deeper tissues thereby affording less recuperative possibility to the overlying skin, or even increased backscattering.

As intimated, the S.E.D varies considerably, excellent observers giving values from 340 to 800 r. Regardless of value given, it must be remembered that the S.E.D is really a physical constant, whereas the skin reaction is subject to all the vicissitudes of any biologic effect. Of all the involved factors, probably the outstanding governing factor of erythema production is the time element, provided the dosage is constant. Schwarz endeavored to formulate the erythema value by devising an intensity exponent for the human skin equivalent to 1.2. Thus, 40 r in 10 min. contrasted to 10 r in 40 min. would be accordingly: $40\ r^{1.2} \times 10 = 836$ r compared to $10\ r^{1.2} \times 40 = 632$ r. The author thinks that Schwarz did not include scattered radiation in his measurements, which might account for his marked discrepancies with other observers. He also wonders at the fact that Schwarz reported an erythema value of 400 r for a very small field. However, if one applies Schwarz's exponent to Holthusen's erythema dose value, viz. 800 r with intensity between 15–50 r per minute, then the following results are obtained: $15^{1.2} \times 53 = 1{,}367$ r S.E.D. and $50^{1.2} \times 16 = 1{,}744$ r S.E.D., as contrasted to the 836 r defined by Schwarz. He points out that Schwarz himself obtained an erythema with intensity of 3 r/min. at 1,700 r; 10 r/min. at 1,400 r; and 30 r/min. at 1,000 r, which with application of his own exponent resolves these values to 2,115 r, 2,212 r and 1,954 r respectively; which is not such a marked discrepancy from Holthusen's figures. He concludes that it is very likely that the true status can be approached by the following formula: $t = \frac{1800}{\mathcal{J}_w^{1.22}}$ with $\mathcal{J}_w$ the working dose per minute.—*W. R. Stecher.*